THE FIELD AND LIMITATION

OF THE

OPERATIVE SURGERY

OF THE

HUMAN BRAIN.

BY

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TO

HIM WHOSE STEPS I FOLLOW,

RICHARD J. LEVIS, A.M., M.D.,

AFFECTIONATELY INSCRIBED.

PREFACE.

My appointment to prepare a paper on Cerebral Surgery for the meeting of the American Surgical Association led to the preparation of this essay. The discussion which it called forth and my reply to the various criticisms upon it may be found in the Transactions of the Association for this year.

J. B. R.

1118 Arch Street, Philadelphia,
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CHAPTER I.

PRINCIPLES OF CEREBRAL SURGERY.

NINE years ago, while resident surgeon in a well-known Philadelphia hospital, I watched a man die after a fracture of the cranium, and found at the autopsy, immediately under the fracture, a cerebral abscess from the surface of which was extracted a piece of bone driven in at the time of injury. I had seen the vigorous man admitted; seen him first intelligent, scarcely sick perhaps; then confused and delirious; then comatose, and finally dead. As the elements of this human life were being successively extinguished by increasing destruction of nerve-centres, the routine and approved medicinal treatment for encephalitis was steadily continued.

It took little surgical experience to recognize the lamentable result of the inactive, so-called conservative, treatment which almost to a certainty sacrificed the patient's life. Do we, Mr. President and Fellows of this Association, know no better at this day than to allow life to be thus destroyed through an almost criminal neglect of the most evident indications for surgical interference?

The recollection of this case and of similar ones has specially directed my attention to the importance of cerebral surgery; and it was with much pleasure that I accepted the President's appointment to prepare for this meeting a paper discussing the operative surgery of the human brain. The design of the paper is to indicate my views in such a succinct and categorical manner that the subsequent discussion may not be discur-

sive. The opinions and practice of the representative surgeons forming this body will thus, I trust, be so formulated that the profession at large may find in its TRANSACTIONS definite rules of action. Dr. Moses Gunn[1] and the present President, Dr. W. T. Briggs,[2] have, at previous meetings, given voice to their belief in more active surgical interference with pathological conditions of the brain. If their words had induced the profession to accept their conclusions, it is probable that I should not appear to-day to reiterate their thoughts.

Since I began the collection of material for this paper there has appeared the exhaustive article of my colleague, Dr. Charles B. Nancrede,[3] which leaves so little to be said, that I was almost inclined to lay aside my pen.

Perhaps, however, constant agitation of the topic may at last gain for those surgical principles which I am about to advocate a wider professional acceptance. It is possible, indeed, that I may take a position rather more radical than that which the authorities mentioned will accept as justifiable. During my remarks I shall report some illustrative cases, but promise that no great amount of time shall be consumed in the relation of their details.

It will, perhaps, facilitate discussion if I at once announce the opinions that I hold upon more or less disputed points involved in the consideration of my subject.

My creed, if I may use the term, is as follows:—

I. The complexus of symptoms, called "compression of the brain," is due not so much to displacing pressure exerted on the brain substance as it is to some form or degree of intracranial inflammation.

II. The conversion of a closed (simple) fracture of the cranium into an open (compound) fracture by incision of the scalp

[1] Treatment of Fractures of the Skull, Recent and Chronic, with Depression, TRANS. AMER. SURG. ASSOC., Vol. I. p. 83.

[2] The Surgical Treatment of Epilepsy arising from Injuries of the Head, with special reference to the use of the Trephine, TRANS. AMER. SURG. ASSOC., Vol. II. p. 101.

[3] Injuries of the Head, International Encyclopædia of Surgery, vol. v. pp. 1–109.

is, with the improved methods of treating wounds, attended with very little increased risk to life.

III. The removal of portions of the cranium by the trephine or other cutting instruments is, if properly done, attended with but little more risk to life than amputation of a finger through the metacarpal bone.

IV. In the majority of cranial fractures the inner table is more extensively shattered and splintered than the outer table.

V. Perforation of the cranium is to be adopted as an exploratory measure almost as often as it is demanded for therapeutic reasons.

VI. Drainage is more essential in wounds of the brain than in wounds of other structures.

VII. Many regions of the cerebral hemispheres of man may be incised and excised with comparative impunity.

VIII. Accidental or operative injuries to the cerebral membranes, meningeal arteries or venous sinuses, should be treated as are similar lesions of similar structures in other localities.

IX. The results of the study of cerebral localization are more necessary to the conscientious surgeon than to the neurologist.

These propositions I shall consider successively:—

I. The complexus of symptoms called "compression of the brain" is due not so much to displacing pressure exerted on the brain-substance as it is to some form or degree of intracranial inflammation.

Many have argued against the use of the trephine in depressed cranial fracture, because considerable depression may exist without interference with the cerebral functions, and because "compression symptoms" frequently disappear after the lapse of a longer or shorter time, on account of the cerebro-spinal fluid, as they put it, gradually becoming displaced or absorbed, and the circulation being restored to its natural condition,[1] and because

[1] H. B. Sands, Annals of Anatomy and Surgery, vol. viii., 1883, p. 104.

deeper lesions beyond the reach of the trephine are frequently found in the fatal cases. Cure in these cases is supposed to occur by adaptation of the brain to the peripheral pressure. On the other hand, writers who favor more frequent operation have also clung to this compression theory, and have advocated perforation of the skull and elevation of the depressed bone for its relief. Their position has been assailed, and, I think, quite logically by those who use the line of argument mentioned above. Neither party seems to see that the so-called "compression symptoms," after traumatism, are not due to compression, but to some form or degree of inflammation. Let the profession repudiate the idea that brain displacement is the pathology of traumatic "compression of the brain," and many of the discordant opinions concerning the utility of trephining will be quickly harmonized. Some writers have, according to Stimson,[1] estimated that the constantly acting intracranial tension under normal circumstances is equal to from eight to twenty-five millimetres of mercury, and susceptible of temporary increase. This tension does, in some instances, overcome the displacement of the cranial vault. Duret,[2] after experimenting with injections of wax, states that a clot amounting to one-twelfth of the cranial capacity, situated between the dura mater and the bone, will cause coma and death in a few hours, but that in the arachnoid cavity a clot one-sixth or even one-fifth the bulk of the cranial cavity is requisite for a lethal effect. This results from the fact that, in the latter case, the wax moulds itself over a large surface instead of being confined to one spot.[3] Lesion of the nerve-centres must, of course, be absent. If this is true, how can any one believe that a square inch of bone, depressed a quarter or a half of an inch, will give sufficient pressure to cause cerebral symptoms, even if the presence of cerebro-spinal fluid does cause the external tension to be communicated to the entire cranial contents? Compression of the brain in cerebral tumors

1 Treatise on Fractures, p. 251.

2 Quoted by Nancrede in International Encylopædia of Surgery, vol. v. p. 64.

3 See Review of Études Expérimentales sur les Traumatismes Cérébraux, by H Duret, in Brain, vol. i., 1878, p. 106.

is the factor that causes many of the symptoms, but I am now speaking of surgical compression symptoms.

There are no definite symptoms by which we can distinguish accurately between laceration of the brain, contusion of the brain, and the so-called compression of the brain. Jonathan Hutchinson is almost correct in thinking that compression of the brain from depressed fragments of broken bone is an imaginary condition.[1] Briggs, with good reason, questions the view that secondary "compression symptoms" in intracranial suppuration are dependent exclusively upon the pressure of the pus. He suggests, and I agree with him, that the symptoms are more probably produced by the inflammatory process which caused the formation of pus.[2] Dr. Hunter McGuire,[3] on the other hand, said, at a recent meeting of this body, "If the fracture is a simple one, and the depression is not sufficient to bring on symptoms of compression, it is better to wait and see if the brain will not be able to accommodate itself to this condition." Here he admits a belief in the pressure theory, though in the next sentence he says that "we all know that there is no relation between the amount of depression and the symptoms of compression." At the same time, Dr. D. W. Yandell[4] gave as his opinion, that we should not elevate the depressed bone unless symptoms of compression are persistent. These opinions were given, I admit, several years ago, and the speakers may have changed their views by this time; but I am sure that to-day such words would still be spoken by many surgeons. The idea that depression of bone causes "compression symptoms" by mechanical pressure is deeply rooted. It would be well if traumatic "compression of the brain" were always translated "inflammation of the brain," and the profession taught to believe it due to irritation of the brain periphery from the traumatic cause.

II. The conversion of a closed (simple) fracture of the cranium into an open (compound) fracture by incision of the scalp is,

[1] Statement of Briggs, Annals Anatomy and Surgery, vol. vii., 1883, p. 65.

[2] Loc. cit., p. 67.

[3] Transactions of the American Surgical Association, Vol. I. p. 92.

[4] Ibid., Vol. I. p. 97.

with the improved methods of treating wounds, attended with very little increased risk to life.

Uncertainty as to the character of a cranial lesion is more dangerous to health and life than the conversion of a closed into an open fracture of the skull, because observation has taught the profession that open cranial fractures do not resemble in fatality similar open fractures of long bones. If I but learn the character of the skull injury, I am acquainted with surgical expedients that render restoration to health more probable than the complication due to the incision renders it improbable.

Antiseptic methods have done away with much of the danger of open-bone wounds, as is shown by the frequent advocacy of resection for ununited and for malunited fractures, and of osteotomy and similar operations for conditions not urgently demanding surgical interference. No surgeon would hesitate to convert a closed recent fracture of the thigh or leg into an open one if it were otherwise impossible to replace fragments which were threatening life. Hence I strongly advocate exploratory incision of the scalp in obscure injuries of the skull.

Some months ago a man, who, it was said, had fallen from a heavy wagon, and had been run over, was admitted into my ward of St. Mary's Hospital. Behind the left ear was a large hæmatoma, where it was asserted the wagon-wheel had struck him. My resident surgeon made a two-inch incision, and removed the clots; but, finding no fracture, closed the wound with sutures, and applied corrosive sublimate dressing. The knowledge that no fracture existed was very satisfactory, I can assure you. Three days later, the incision had healed up without suppuration except in a space of three-eighths of an inch, and the sutures were removed. Such rapid union without complication will not always occur, but it shows the possibility of little risk in the majority of cases.

I recently ligated the brachial artery, and never dressed the wound after the hour of operation, because it was healed when the dressing was first removed. Such results of antiseptic surgery make us properly bolder than our predecessors, who feared to convert a closed fracture of the skull into an open one.

III. The removal of portions of the cranium by the trephine or other cutting instruments is, if properly done, attended with but little more risk to life than amputation of a finger through the metacarpal bone.

Much of the mortality attributed to trephining belongs to the serious brain-lesions that have accompanied the fractures for which trephining has been done, and to the absence of proper surgical antisepsis. Many patients have been trephined and have undoubtedly died; but the opponents of trephining must show that cause of death lay in the operation itself. In deaths occurring from lesions for which trephining is the admitted treatment, they must likewise show that the operation was done early enough to remove the causative factor of death before they can assert that the operation was unavailing. Gross, Michel, and many others, have frequently quoted historical facts and cases which show the slight risk incurred by uncomplicated trephining. Briggs says, "My opinion, based on a large personal experience, is, that trephining the skull is one of the safest of the capital operations of surgery."[1]

The opposite view, however, was held by Dr. H. F. Campbell, at a recent meeting of this Association, at which he said: "I have ever regarded trephining as one of the most serious of all capital operations."[2]

Nancrede,[3] from a careful investigation, gives a mortality of 10.69 per cent. as being a probably fair estimate of the risk of the operation *per se*, and a death-rate of 15.29 per cent. as an expression of the probable risk in trephining a simple depressed fracture. This author further says that his own experience has taught him that trephining is not a dangerous operation, and that more patients die from complications, that might have been prevented by timely operation, than from the removal of a disk of healthy bone.

Dr. R. W. Amidon, of New York, has collected 115 cases of trephining and kindred operations occurring since 1879. These

[1] Annals of Anatomy and Surgery, vol. vii., 1883, p. 65.

[2] Transactions of the American Surgical Association, Vol. I. p. 94.

[3] International Encyclopædia of Surgery, vol. v. pp. 94, 95.

operations were done for various causes, and were unselected by Dr. Amidon; nor did he confine himself to cases treated antiseptically.[1] Of these 115 unselected cases, 29 died; but of these, 25 presented, at the time of operation, symptoms endangering life, leaving therefore but 4 cases in which the fatal issue could be attributed to the operation. This gives a mortality of a little over 3 per cent. to the operation. He announces his reasons for considering the 25 deaths as not attributable to operation in the following words: "In six cases, symptoms of abscess of the brain declared themselves before the operation was performed. In five a meningitis existed at the time of operation. In four cases shock caused death; two died of hemorrhage from a branch of the middle meningeal artery (not wounded in the operation); one died of hemorrhage from the middle cerebral artery, severed by a stab wound of the head; one died of hemorrhage from a lacerated longitudinal sinus; one of galloping consumption, which was hereditary; one of pneumonia; one of extensive laceration of the brain; one of opium poisoning; and three I accept, on authority of the physician reporting them, as not dying from the effects of the operation."[2]

Yeo[3] trephined 26 monkeys under antiseptic precautions, and had only one death attributable to intracranial inflammation, though six other deaths from exposure to cold weather, chloroform poisoning, or hemorrhage occurred among his cases. In some of the animals portions of the brain were excised. Of other monkeys trephined without antiseptic precautions all died.

Much stress has been laid by some writers on the danger of wounding the membranes and brain with the trephine. With a conical trephine or the burr of the surgical engine, as recommended by Dorr and by myself,[4] there is no danger of this.

[1] Annals of Surgery, St. Louis, March, 1885, p. 205; see, also, his previous paper, Medical News, June 21, 1884.

[2] This list accounts for the death of 26 instead of 25 patients, and apparently contains an error.—J. B. R.

[3] British Medical Journal, May 14, 1881, p. 763.

[4] Buffalo Medical and Surgical Journal, xix., 1879–80, p. 475; Phila. Med. Times, 1881–82, xii. 206.

Even if such an accident should occur to the membranes, it is, in my opinion, of very minor importance unless the damage is much greater than could occur except by gross carelessness. Dr. Gunn disapproves of the use of the mallet and chisel, because he believes that the repeated shocks to the brain may prove injurious to the nerve-tissue.[1] For removing large or irregular areas of bone, the flat burr of the surgical engine is certainly much more accurate and desirable.

I have compared trephining with amputation through the metacarpal bone, because in both operations there is exposure of cancellated bone structure. I do not know that the mortality of such finger amputations has been accurately computed, but is certainly regarded by all as slight. It is seldom that patients are confined to the house after such amputations. Trephining in itself is, I am convinced, little if any more hazardous. I believe that one of us trephined to-day might, if it were necessary, go home without incurring any great risk to life; though I would not advise such a procedure. Amputation of the finger may be followed by erysipelas, septicæmia, or death; so may trephining, but it is not to be expected. The mortality of amputations of the thumb and fingers is, according to Ashhurst,[2] 3.3 per cent. This undoubtedly is less than the mortality of amputations through the metacarpus, because amputations of distal phalanges, which are almost without risk, are of course included. The same writer gives partial amputations of the hand a mortality of 6.6 per cent. If, therefore, we estimate amputation of a single finger through the metacarpal bone as having a mortality of 4 or 5 per cent., it will probably be nearly correct. According to the figures of Amidon, given above, trephining is actually much less dangerous to life than this.

IV. In the majority of cranial fractures, the inner table is more extensively shattered and splintered than the outer table.

Many experimental fractures made in the dissecting-room, and observation of cases in the practice of myself and of others,

[1] Transactions of the American Surgical Association, Vol. I. p. 88.

[2] International Encyclopædia of Surgery, vol. i. p. 637.

teach me that extensive shattering of the inner table, with only a moderate amount of fracturing of the external table, is of frequent occurrence in other as well as in punctured fractures. I admit that the condition in the cadaver, preserved by zinc chloride, with its shrunken brain, is different from that in the living; but there is much evidence of the same splintering to be found in the study of accidental and homicidal cranial fractures. This is in accordance with the well-known mechanical law, that compressing force applied to the outside of a surface, as are undoubt-

Fig. 1.

Outer Surface of Fractured Skull.

edly most fracturing forces applied to the skull, tends to produce more extensive breaking of the inner surface. This is especially so in all localized blows. Punctured fractures have long been treated by early trephining, to avert encephalitis. For the same reason I recommend resort to trephining even in more diffused and less accentuated fractures. It is to prevent inflammatory sequences due to splinters forced into the membranes and brain, and to avert the consecutive occurrence of epilepsy and insanity, that the operation should be performed; not because of the

fear that symptoms of compression of the brain may arise, nor because necrosis of detached portions of bone may occur.

Sometimes there is no fissure in the outer table, though the inner table is extensively broken and depressed. Twenty such cases are reported as having occurred during the late civil war.[1] All of these patients died from intracranial inflammation except one, in which the splintered portion of the inner table was removed as a sequestrum.[2] I show you a piece of skull removed from a patient who recently died under my care.

Fig. 2.

Inner Surface of Fractured Skull.

He was struck with a pitcher, which caused a small scalp wound through which my finger-tip felt rough bone. I enlarged the incision, came upon a very rough surface, due to unusual irregularity of the lambdoidal suture, with small Wormian bones, and found only a small dent or fissure looking much like

[1] Med. and Surg. Hist'y Reb., Pt. I. Surgical Volume, p. 150.

[2] Prescott Hewett and Lidell have furnished other statistics of such cases. Holmes's System of Surgery, Am. edition, 1881, vol. i. p. 636.

the entrance for a vein. I determined to do exploratory trephining because of the nature of the vulnerating force. Dr. George Dock, under my direction, cut out a disk of bone close to the external dent, at the position which was thought would give best access to any splinters. Nothing was found but a small fissure crossing the inner surface of the disk. A probe slipped between the inner table and the dura disclosed no irregularity; therefore no further operative steps were taken. A portion of the inner table left in the bottom of the trephine-hole was undisturbed because it was smooth. The patient died in about forty-two hours of delirium tremens. Dr. H. F. Formad, the pathologist, found no inflammation of brain or meninges, but intense œdema of the brain and membranes, and at the inquest swore that death occurred from alcoholic delirium. The section of bone presented to you shows a marked depression of the table due to a T-shaped fracture under the seat of the external dent, beneath which was a small clot upon the dura mater. The top line of the T, which in the figure is vertical, is $1\frac{1}{4}$ inches long. The cleansed bone shows a semi-elliptical fissure of the external table encircling the slight dent. This was not seen at the time of operation, because the surface at that distance from the wound was left covered by periosteum. It would have been better if the trephine had been applied directly over the external dent instead of alongside of it. It is well, therefore, to follow that well-known rule in such cases as this, and also to employ a large trephine. Where a mere entrance for the elevator is desired, a small trephine should be used.

If this patient had lived, he would have been very liable, I think, to epilepsy or insanity. Only a few weeks ago I saw, in consultation with Dr. Charles K. Mills, a man of 22 years who was suffering with marked mental impairment occurring, as he and his brother said, subsequent to a fracture of the skull received about four years previously. Reference to the notes of the Pennsylvania Hospital, in which he had been treated, showed that he had been admitted for a compound depressed fracture of the skull, which was so slightly marked that operation was not deemed necessary. It is probable that his mental

impairment was due to a depression similar to that seen in this specimen. It was my intention to trephine in this case of mental failure if further investigation of his condition by Dr. Mills conclusively traced the aberration to the injury. Unfortunately, the patient passed out of our control by returning to his country home before I had a second opportunity of examination.

I feel sure that the element of danger in skull fractures is this splintering of the inner table; and I differ most decidedly from those who esteem it of comparatively little importance. Its danger in those abruptly depressed fractures, called punctured fractures, has been quite generally recognized; but its great frequency and risk in other forms of fracture are still not sufficiently emphasized by all authorities. Ashhurst, in the last edition of his Surgery, dated 1882,[1] says, in speaking of simple depressed fractures, "I have never seen a case of this kind in which I thought the use of the trephine justifiable, nor an autopsy which showed that the operation could possibly have saved life." In compound depressed *impacted* fractures he would not advise operation, "even if symptoms of compression were present." He goes on to say that the trephine "is not to be used with the idea of relieving compression, nor with the idea that there is any special virtue in the operation to prevent encephalitis." In punctured fracture the same distinguished surgeon thinks the trephine may be necessary to slightly enlarge the opening in the skull to remove the spicules which are apt to be broken from the more extensively involved internal table; but says, "It is better to leave imbedded in the brain, a foreign body, or even a fragment of bone, than to add to existing irritation by reckless attempts at its removal."

Perhaps Dr. Ashhurst has changed his views within the last two years. If he has not, I must disagree with him on this subject, except in the opinion that reckless surgery is always unjustifiable. His definition of what constitutes reckless surgery may differ from mine.

[1] Pp. 325, 326.

Many writers, while admitting the probability of greater damage to the inner table, do not give its disastrous consequences sufficient stress. Briggs,[1] however, truly says that the great danger in depressed fracture is not compression but inflammation set up by displaced fragments of bone. Dr. Sands says[2] that advocacy of early trephining, on the ground that loose pieces of bone in simple comminuted fracture will probably become necrosed and set up fatal intracranial inflammation, is improper. I believe that few advocates of early trephining give necrosis as a reason for their belief. Necrosis is not, but encephalitis is, very liable to occur.

Dr. Sands also believes that the apprehensions felt by those who advocate preventive trephining in closed depressed fracture without head symptoms, with the object of removing sharp fragments of the inner table, are scarcely justified by observation. These extensive osseous lesions are, in his opinion, often recovered from without surgical interference. He states[3] that immediate resort to the trephine is imperatively required, however, in fractures of limited extent, and in those in which there is reason to think, from their situation or the occurrence of monoplegia, monospasm, or hemiplegia, that a splinter has penetrated the motor area of the cortex. Trephining is also demanded, in Dr. Sands's opinion, when compression of the brain is due to blood between the dura and cranium.

V. Perforation of the cranium is to be adopted as an exploratory measure almost as often as it is demanded for therapeutic reasons.

I have shown that the occurrence of fatal encephalitis is frequently due to spiculation of the inner table, and that spiculation or extensive shattering of the inner table is common in limited fractures of the external table. Hence it follows that exploratory perforation of the cranium is justifiable in all cases where the nature of the impinging force, or the appearance of

[1] Annals of Anatomy and Surgery, vol. vii. p. 69.
[2] Ibid., vol. viii. 1883, pp. 101–103. [3] Ibid., p. 106.

the external table, renders spiculation of the inner table probable, provided that less danger to life and health is inherent in perforation than in the probable spiculation. I have already asserted my belief in, and given reasons for, a low mortality risk of perforation. I am of the opinion that fractures of the cranial vault, produced by such general application of force as occurs when a man falls from a great height upon his head, are less frequent than fractures by direct and comparatively localized blows, such as occur from ordinary missiles, bullets, and falls from low elevations. These latter are those which tend to produce internal spiculation. Hence I am driven to the conclusion that exploratory perforation to determine the absence or presence of internal spiculation is often demanded by the uncertainty of the invisible condition. Without a knowledge of the true state of affairs treatment is empirical; and the risk to subsequent mental health or to life is too great to permit reliance on empirical treatment, when a knowledge of the true condition is obtainable with the slight danger that pertains to antiseptic trephining.

Whenever the fracture, whether originally an open one or so made by an incision, presents the possibility of the inner table being detached and splintered more extensively than the outer, I should be inclined to advise perforation. In other words, I would cut the scalp to see the condition of the outer table, and I would cut the bone to see the condition of the inner table, in every case where the risk of obscure knowledge is greater than the risk of divided scalp and perforated bone.

The tendency to procrastination in such matters has destroyed many lives. Nancrede[1] recommends early preventive trephining strongly, because, after encephalitis has once begun, trephining does not remove the inflammation, but merely one source of irritation without influencing the existence of the inflammatory process which has been aroused. His statistics show the mortality of the operation, after symptoms of brain disease have arisen, to be much more than twice as great as in preventive operations—the figures are 52.8 per cent. and 22 per cent. He

[1] International Encyclopædia of Surgery, vol. v. p. 95.

further says, that, although the operation should be done early, it is never too late in neglected cases to make the attempt; for an abscess may be found and evacuated with the result of saving life. Stimson[1] says that the percentage of recovery in early operative interference is actually high compared with tardy operations; and cites instructive cases to prove that his opinion is correct. Wound of the longitudinal sinus and removal of about three square inches of bone were no bar to recovery in a case which he treated by immediate trephining, though no brain symptoms except stunning were present.

VI. Drainage is more essential in wounds of the brain than in wounds of other structures.

That drainage is as essential in wounds of the brain as in wounds of other structures would seem to be a self-evident proposition; yet gunshot and other wounds of the skull and of the cerebral membranes and hemispheres have long been treated as if drainage was not a necessary factor in therapeutics. A surgeon who would think himself censurable to leave the opening leading into a suppurating cavity obstructed by shreds of skin and fascia, will, with profound equanimity, see his patient die with pus burrowing among the vital centres of the brain. There may be but a small fissure or opening, or perhaps none at all, in the bony wall inclosing the brain, and but a mere puncture in the tense dura mater, yet he will hesitate to give free exit to the pus. We split open a knee-joint, we incise a pleural, peritoneal, or pericardial cavity to give free egress of pus; so must we cut away the cranium and incise the dura mater in analogous conditions. The greatest error in this direction has, perhaps, been in an expectant, instead of an operative, treatment of bullet wounds of the brain.

Stimson,[2] in speaking of the trephine, says, "Most writers upon surgery during the last twenty or thirty years condemn its use unequivocally, except in compound fractures with depression and with marked and persistent cerebral symptoms."

[1] Treatise on Fractures, p. 248.

[2] Ibid., p. 247.

A very few writers could be mentioned who are not included in the class spoken of by Stimson, but who have advocated more frequent use of the trephine in such skull fractures as are seen in civil practice. The hesitation to employ the trephine in bullet fractures, however, has been almost universal.

This is, I think, very erroneous doctrine, for death from abscess is very common after bullet wounds of the brain. The fracture is compound, the suppurative process recognized as almost certain to occur, and yet no provision of moment has, according to the usually pursued treatment, been made for drainage and antisepsis.

In a considerable number of open linear fractures of the cranium, pus has been found at the autopsy beneath the bone or membranes. The danger of an early trephining in such cases would have been less than that resulting from the imprisoned pus. The mere line of fracture establishes a direct communication between the air and the membranes which may lead to suppuration. Such communication is, of course, much more extensive in gunshot fractures and comminuted fractures. In cases, therefore, where there is reason to believe that suppuration will occur, trephining may be beneficial for purposes of drainage. I do not advocate perforation in all linear fractures which are not subcutaneous; but advise it when there is such strong probability of suppuration as is the case in nearly all gunshot fractures.

The statistics of Dr. H. R. Wharton,[1] concerning foreign bodies lodged in the brain, show the importance of removal when possible. Of 106 cases in which removal was accomplished only 34 died; of 210 cases in which no attempt at removal was made 122 died. The death-rate here was doubtless largely due to want of proper drainage.

While preparing this paper I assisted Dr. R. J. Levis in a case which will illustrate this phase of my communication.

A young man of nineteen years was shot in October, 1884, with a pistol of 22-hundredths calibre, held about a foot from his head.

[1] Philadelphia Medical Times, July 19, 1879.

The ball entered the skull at a point about $1\frac{1}{2}$ inches behind and $2\frac{1}{4}$ inches above the cartilaginous attachment of the left ear, taking a direction slightly downward. He fell senseless, but soon reacted. The next day when seen he had no fever, no palsy, no strabismus, no abnormality of pupils, and no intellectual obscurity. Towards evening, however, his sight began to decrease, and by the morning of the second day after the accident he was totally blind, and had no pupillary reaction to light. Dr. A. H. McAdam asked Dr. Levis to assist him in the treatment of the case. A probe carefully inserted into the skull entered for a distance of about $3\frac{1}{2}$ inches. The following afternoon (3d day) Dr. Levis trephined for the purpose of affording free drainage and of attempting extraction of the bullet, which he thought that he possibly had felt with the probe. An opening was made, the membranes incised, and several spicules of bone extracted from the cerebral hemisphere into which they had doubtless been driven by the ball. The brain-substance was already so broken down that it oozed out before the disk of bone was cut entirely loose, and the full length of my little finger was readily carried into the brain wound. The ball, not being readily found, was not searched for very long. On the next day (4th) the patient could distinguish the hand passed before his eyes. Two days later he could see to tell time approximately by a large clock hanging across the small room.

After the lapse of two more days, according to the notes of the case, he could tell the minutes indicated by the clock, which had a dial about ten inches in diameter, and was about six feet distant from him. Vision seemed better in the left than in the right eye, and when looking he closed the right eye as if some diplopia existed. Free suppuration from the wound continued, no marked febrile action occurred, and subsequently he became able to tell time by a watch. Nine weeks after the injury there occurred an abscess at the site of the trephining, but three months subsequent to the shooting he was attending to his business in good health, though the wound had not yet cicatrized. He could see well. I saw the patient only at the time of the operation, and have taken these facts from notes obtained by Dr. C. L. Bower.

I examined this patient in April, 1885, six and a half months after injury. He was in good health, weighed twenty pounds more than usual, drove a wagon, which he loaded with baskets and bundles, and had as good memory as ever for numbers, names, and words.

No failure in this respect was noticed by him in his occupation of delivering goods. He had no headache, and no tenderness or pain at the cicatrix of the scalp. I found that his left ear heard my watch tick at 24 inches, while his right heard it at 42 inches. This defect in hearing he had observed, and seemed to attribute it to the injury. He whistled well, but protruded the tongue with its tip a little to the left. No loss of tactile sense did I discover in testing sensation of the fingers with the points of a pair of compasses. He never observed trouble in vision before the injury, but said that now he could not read the newspaper well. The pupils reacted to light, and were neither dilated nor contracted. The right eye showed vision $\frac{20}{c}$, not perceptibly improved by spherical lenses or the stenopæic hole; the left eye $\frac{20}{LXX}$, not perceptibly improved by spherical lenses or the stenopæic hole. He read D 1.25 at $4\frac{1}{2}$ inches with difficulty, which convex lenses did not remove to any marked degree. No diplopia was noticed by him when looking at lights or stars. No strabismus, and no insufficiency of the internal rectus muscles were observed by me. There was, however, hemianopia of both eyes, when a pencil was held to the right of the middle line of each eye. The sense of smell in each nostril was apparently unaffected, for he easily distinguished carbolic acid from Cologne water. On each side of the tip of the tongue he readily distinguished tannin from chloride of ammonium. I regret that I had not a good opportunity of examining the ears and eyes with the ear mirror and ophthalmoscope respectively.

While admitting that a similar case might have recovered without operation I doubt its probability; the spicules driven into the brain, the softening therein already begun, and the profuse suppuration through the perforation, make me believe that death from intracranial abscess would have occurred rapidly if no operation had been done.

Agnew[1] quotes MacLeod as saying that in the Crimean War, of 67 gunshot fractures of the skull in which the skull was penetrated, one hundred per cent. died. Agnew himself gives a table[2] of 486 cases of penetrating gunshot fracture, with a mortality of 85.5 per cent. These high death-rates are largely,

[1] Principles and Practice of Surgery, vol. i. p. 287. [2] Ibid., p. 288.

I believe, due to inefficient drainage; but also to splintering of the inner table and driving of the splinters into the brain-substance. Dr. S. W. Gross is quoted by S. D. Gross as showing in over seven hundred cases of gunshot wounds of the skull only 25 + per cent. of recoveries by expectant or conservative treatment, but 41 + per cent. of recoveries after operative treatment; or a difference of about 16 per cent. in favor of operative interference. These statistics of Gross[1] were published eighteen years ago, and would probably be improved by the present operative methods.

Drainage of cerebral abscesses and wounds by tubes or horsehair has been successfully performed on several occasions. Noyes[2] passed a drainage-tube through a traumatic abscess in the frontal lobe, utilizing the trephine hole and the orbit for its entrance and exit. In this case death occurred. The four cases of similar operative treatment, reported by Burchard, by Fluhrer,[3] by Fenger and Lee,[4] and Kemper,[5] all recovered. Dr. J. P. Thomas,[6] of Kentucky, reports a case of cerebral abscess, which he successfully treated in 1875 by drainage obtained by position and the use of tents made of strands of silk.

These cases are sufficient to prove the truth of my sixth proposition.

VII. Many regions of the cerebral hemispheres of man may be incised and excised with comparative impunity.

Physiological experimenters know well that the lower animals bear, without serious risk to life, ablation of quite large areas of the brain-substance. Although clinical experience has shown the same fact respecting the human brain, surgeons have not

1 System of Surgery, ed. 1882, vol. ii. p. 71, from American Journal of Medical Sciences, October, 1867.

2 American Journal of Medical Sciences, July, 1882.

3 Medical News, January 10, 1885, and New York Medical Journal, March 28, 1885.

4 Transactions of the American Surgical Association, Vol. II., 1884, and American Journal of Medical Sciences, July, 1884.

5 American Journal of Medical Sciences, January, 1885.

6 Medical News, February 14, 1885.

been sufficiently impressed with the circumstance. They have looked upon the brain and its membranes very much as until recently they looked upon the heart and pericardium—regions never to be approached with trocar or scalpel. Many patients have died of brain abscess or brain tumor, because the timidity of the surgeon prevented the introduction of an aspirating needle or knife to open the pus cavity, or the employment of enucleation for the detachment of a neoplastic growth. This is partly due to the ignorance of surgeons respecting the localization of cerebral functions.

Nancrede[1] reports a remarkable case in which a patient, who was thought to be dead from cerebral abscess following penetration of the skull by a knife, was restored to conciousness by a second trephining and incision of the brain-substance. An aspirator needle had previously been passed several times into the brain to the depth of two-thirds of an inch through the first trephine hole, but no pus had been found. After the incision had evacuated one or two fluidounces of pus, the man revived and lived till the sixth day.

Dupuytren, Detmold, J. F. Weeds, Hulke, and others have reported recoveries after incision of the brain and evacuation of abscesses. Nancrede finds out of thirty cases of cerebral abscess treated by operation a mortality of only 50 per cent. I do not know in how many of these cases the brain-substance was incised or punctured in the search for pus.

Amidon[2] states, that of 100 cases of operation on the head, collected by him during recent years, the dura mater was opened in 33 by either the injury or the surgeon. These gave a mortality of 39.3 per cent.; but he states that in only one death could the fatal issue be directly ascribed to the exposure of the cerebral convolutions, and therefore he gives the mortality of operation inducing exposure of the hemispheres of the brain as 7.6 per cent. As showing the tolerance of the brain-substance to injury, he quotes among others the following recent cases, of which he gives the various sources as below mentioned.

Fluhrer's case. A bullet entering at a point about four centi-

[1] International Encyclopædia of Surgery, vol. v. p. 83; and TRANSACTIONS OF THE AMERICAN SURGICAL ASSOCIATION, Vol. II., 1884.

[2] Medical News, June 21, 1884.

metres above the left eye, a little to the left of the median line, pierced the first frontal convolution, traversed the brain backwards and a little outwards, and emerged from the upper part of the parietal lobule, where it lay slightly imbedded in the brain. The symptoms were not alarming. The course of the bullet was ascertained by careful probing, and the trephine was applied over the supposed site of the ball. After a short search it was found, and a drainage apparatus composed of horsehair and catgut passed through the entire track of the ball. The operation was done with antiseptic precautions, and the wounds were dressed with iodoform. Recovery was uninterrupted and complete. The slight weakness of the right hand and loss of memory rapidly disappeared.[1]

Olpherts's case.[2] The whole of the right parietal, parts of the right occipital, temporal, and left parietal bones were torn away with extensive laceration and loss of cerebral substance. The case was treated antiseptically without operation, and recovered perfectly.

Wood's case.[3] A man was struck by the pilot of an engine, and his head cut open from the inner canthus of the left eye to the occiput. A furrow eight centimetres long was cut in the anterior lobe of the left hemisphere. Under cold applications he recovered.

Hoskins's case.[4] A boy six years of age was stepped on by a horse. The left side of the head was crushed in, blood, bone, and brain escaping. Blood and portions of brain exuded from left ear. It was estimated that 62 c.c. of cerebral substance were lost. There were three compound fractures. There was a right hemiplegia which disappeared, and an aphasia which improved.

These cases certainly prove the truth of my seventh proposition, which would also seem self-evident from the occasional continuation of life and even of comparative health during the existence of large areas of brain softening or suppuration, or of large substitutive growths in the cranial cavity.

1 This case, quoted by Dr. Amidon with permission of Dr. Fluhrer, has since been published with most valuable remarks in the New York Medical Journal March 28, 1885.

2 Olpherts, British Medical Journal, 1882, ii. 89.

3 Wood, American Journal of Medical Sciences, July, 1881, lxxii. 168.

4 Hoskins, Lancet, 1883, ii. p. 99.

VIII. Accidental or operative injuries to the cerebral membranes, meningeal arteries or venous sinuses, should be treated as are similar lesions of similar structures in other localities.

Numerous cases have recently been reported that prove this assertion. W. T. Bull[1] obtained good results in two cases after suturing the divided dura mater with catgut. Parkes[2] had a cure following lateral suture of the superior longitudinal sinus. W. B. Hopkins,[3] of Philadelphia, recently treated successfully a profuse hemorrhage from the superior longitudinal sinus, after removing comminuted bone, by pressure with a pad of lint sprinkled with iodoform. Sands[4] applied pressure to a wounded superior longitudinal sinus. Brinton[5] has applied lateral ligature successfully to the lateral sinus. Nancrede[6] has successfully ligated the middle meningeal artery. Numerous other cases of this sort might be cited, but enough have been quoted to prove my proposition.

IX. The results of the study of cerebral localization are more necessary to the conscientious surgeon than to the neurologist.

If any of the cerebral functions are with certainty recognized as located in definite convolutions and regions of the cortex of the cerebral hemispheres; if it has been demonstrated that perforation of the skull and incision of the membranes and brain will at times prevent the destruction of life by intracranial inflammation, abscess, or tumor; then it follows as a direct conclusion that a man, who assumes the rôle of a specialist competent to relieve disease by operative measures when medicinal therapeutics become impotent, must, if he is conscientious, make the subject of cerebral localization an object of study. I hold that such study is more necessary to him than to the neurologist, though the latter may, and probably does,

[1] Archives of Medicine, vol. i. 1879, p. 219.
[2] Annals of Anatomy and Surgery, viii. 1883, p. 118.
[3] Annals of Surgery, July, 1885.
[4] Annals of Anatomy and Surgery, vol. viii. 1883.
[5] Philadelphia Medical Times, vol. xii. 1881, p. 577.
[6] Ibid., March 8, 1884, vol. xlv. p. 440.

need a more detailed knowledge of the subject. The surgeon, when called upon to decide as to the propriety of operation, is often, and, indeed, usually, the sole authority by whom the question must be determined. If operation is required in these cases, it is required at once; no time can be wasted in waiting for the services of a neurologist living in a distant part of the county or State. Often, time to consult even books is scarcely available. Therefore, as many cerebral functions *have* been definitely localized, and many cases *have* shown that life can be saved by perforation of the skull and incision of the membranes and brain, I hold that the study of cerebral localization *is* more important to the conscientious surgeon than to the neurologist, who is seldom called upon to determine such questions in haste, except when the surgeon finds his own knowledge deficient.

Reference to a few of the cases in which cerebral localization, or functional cerebral topography, has been well utilized in surgical practice is here proper. Afterwards, I shall detail some of the facts which experimental and clinical investigation have proved to be of value to surgeons.

The recent removal by Godlee[1] of a gliomatous tumor the size of a walnut from beneath the gray matter of the upper part of the ascending frontal convolution, has probably attracted the notice of you all. The patient died after the expiration of four weeks from meningitis, which it is possible may have been due to inefficient drainage of the wound, as I have seen no statement that drainage was provided.

The chief symptoms which led Dr. Hughes Bennett, who had charge of the case, to diagnosticate the extent and locality of the tumor were paroxysmal twitchings of the left side of the face, alternating with twitchings of the left hand and arm, followed by slowly progressive paralysis of the hand, and later on by twitchings of the left leg and eyelid, and paresis of the left leg. These symptoms were accompanied by double optic

[1] Lancet, December 20, 1884, and January 3, 1885; see, also, Annals of Surgery, March, 1885, for Dr. Amidon's review of the case.

neuritis and violent headache. Dr. Bennett concluded that there was a brain tumor involving the cortical substance of the right hemisphere; that it was probably of limited size, as it had destroyed the centres presiding over motion of the hand, but had not yet destroyed the centres of the leg, face, and eyelids; and that to reach it, trephining should be done at the upper third of the fissure of Rolando. The operation was done, and the tumor readily removed. The lancinating pains in the head, the vomiting, the twitchings of the limbs were relieved; the temperature was, up to the twentieth day, never over 100°, and the pulse not above 90; and the patient was perfectly intelligent. Death occurred on the twenty-eighth day after rigor, fever, and pain had occurred during several days preceding.

It has been stated[1] that Macewen, of Glasgow, has twice successfully removed foreign growths, of which the site had been determined by the principles of cerebral localization. Other successful operations for localized purulent and hemorrhagic exudations, similarly detected, have been attributed to the same surgeon. He recently wrote me that he had not yet put the records of his cases in print, and that the accounts which had already appeared of some of them were not very trustworthy; but that he shortly intended to publish all of his cases bearing on this point. We are still, therefore, without any authoritative statement of the details of his operations. Broca, Lucas-Championnière, E. C. Seguin,[2] and E. C. Fuller[3] have given careful attention to, and valuable clinical information upon, the subject of cerebral topography in its surgical relations.

Dr. M. Allen Starr[4] has given a valuable analysis of American cases, in which there existed localized cortical lesions of the brain. Some of the cases are surgical, others medical, but his two papers deserve the careful perusal of every surgeon.

The following case of operation from the unpublished notes of Dr. Chas. K. Mills shows the value of cerebral localization:—

[1] Boston Medical and Surgical Journal, January 15, 1885.
[2] Archives of Medicine, December, 1882.
[3] Ibid., vol. ix. p. 262, 1884.
[4] American Journal of Medical Sciences, April and July, 1884.

Epilepsy following Cranial Traumatism; Situation of Meningeal Lesions diagnosticated by the Principles of Cerebral Localization; Trephining; Death.—A man, aged about 40 years, without any personal venereal history, and belonging to a family without any epileptic history, had, it was said, excellent health until eleven years before he came under Dr. Mills's observation. At that time, in 1873, he was struck upon the head many times with a boot-jack, and the mother was told by a physician that the skull was crushed, though the skin was not lacerated. Consciousness was not lost at the time of the injury. Great swelling of the parts followed immediately, and there occurred intense headache, which was almost constant, though much worse at times than at others. This was soon succeeded by attacks of vertigo, which sometimes resulted in his falling to the ground; consciousness, however, was retained. These attacks increased in severity until about three years after the receipt of injury, when he first began to suffer with true epileptic paroxysms, chiefly petit mal. The convulsions were slight or wanting, and unconsciousness lasted only a short time. The mother states that once a month he would have from three to six fits, after which he would be exempt from them until the next change of the moon.

The seizures gradually increased in severity and number during the eight years following his first paroxysm. During all these years he suffered with headache, which was always located at the seat of injury, which at times was so intense as to cause him to throw his hand to the seat of pain, and run and bury his head in his mother's lap.

About the time he first began to lose consciousness during the attacks (three years after injury) his eyesight rapidly began to fail, and he entered the Wills Eye Hospital, where he remained a short time. Previous to his death he was only able to see the name of the paper on a newspaper, and partly on this account, as well as on account of his headache, he seldom went out unattended by his mother. During the past weeks he appears to have suffered more than usual from his headache, which was described as a throbbing at the seat of the former injury, and his epileptic paroxysms came on him more frequently and were of a more convulsive character. His mother states that he would never bite his tongue during the convulsions, but that there would be some frothing at his mouth, and that his jaws would be fixed.

Five or six days before entering the Philadelphia Hospital, he

first noticed a weakness of the right arm, and a little later of the leg on the same side (the seat of injury being the left). About two days later he was unable to raise his arm, and in walking dragged his right leg. His mother does not know whether there was a disturbance of sensibility on the affected side or not.

His mother states that the convulsions followed each other at intervals of about half an hour, and that between them his mental condition had been the same as it was when Dr. Free, resident physician, found him on arriving in the ambulance. He was at that time lying on the floor, almost nude, in a maniacal condition, constantly repeating "I did not do it," all the while throwing his left hand up over the left side of his head, and then again down to his side or over his abdomen. His right arm and leg he did not move. The jolting of the ambulance seemed to promote his paroxysms, as he would have them every five minutes. On arriving at the hospital he was given 80 grs. of bromide of potassium, but with no effect. Nitrite of amyl was tried, but was found equally useless. Then inhalations of ether during the paroxysms, with hypodermic injections of morphia and atropia, were administered, which shortened the attacks. During the convulsion the left arm was in a state of rigidity and the fingers clinched. There was also marked clonic spasm of the arm and leg of the right side, alternating with some rigidity. The spasm in the right side differed from that in the left in being both clonic and tonic. The eyes were fixed, deviating to the right side, and the pupils very much dilated, the left slightly more so than the right; especially was the latter fact noticeable between the paroxysms. In the intervals his eyes showed a beautiful example of horizontal oscillation. This was bilateral, and the eyes acted together with perhaps a slight internal strabismus. Pulse very irregular, ranging from 100 to 160. His convulsions during the night followed each other at regular intervals of from 13 to 15 minutes, and if they were anticipated by inhalation of ether would be postponed until the end of the next 15 minutes.

About midnight he began to use his arms less wildly, his paroxysms occurred at longer intervals, and his pupils, which before had been widely dilated, contracted almost to pin-head size, but dilated during the paroxysms. Respiration was impeded or almost arrested after the convulsions, but never became stertorous. He passed very little urine. He became comatose, and remained so until June 5th, 5 P. M.

At this time the patient was first seen by Dr. Mills, and in consultation with Dr. Hearn it was decided to trephine. Although the mother stated that the injury was on the left side of the head, well forward, it was not indicated by any scar; but Drs. Mills and Hearn decided to trephine on the left, about the Rolandic line, concluding that the convulsions on the left were due to involvement of the dura mater, while those on the right to cortical irritation of the left hemisphere, in addition to the dural spasms.

The operation was done without the aid of an anæsthetic, and a piece of the skull removed. The dura mater was found rather more injected than was normal, and slightly adherent to the portion removed. No depression or fracture of the internal table was apparent. Soon after the operation he became quieter, his convulsions less marked, and he was again able to swallow and take a little nourishment during the night.

On the morning after the operation he spoke a few words intelligibly, and recognized his mother.

Nourishment was given him both by stomach and rectum, as he could retain but little in his stomach. His convulsions during the day and night were slight, and involved both sides. June 7th and 8th, second and third days after operation, it was evident that the man was becoming weaker, his pulse more feeble and rapid, but his temperature not much above normal; his breathing was hurried, but not stertorous, and he slept a considerable portion of the time. On the second day he had about six convulsions and but four on the third. On the night of June 8, 80 hours after the operation and four days and a half after beginning of his attack, the man died without a struggle, having had no convulsion during the night.

Autopsy held 13¾ hours after death.

Scalp wound the result of trephining, which had been performed June 6, 1884.

On the left side, at the seat of operation, the dura mater of convexity of left hemisphere, at a point one-fourth inch in front of fissure of Rolando and forward, was firmly adherent to pia mater and substance of the brain. The tissues as a whole formed a common mass. The dura mater was adherent to the skull over the anterior portion of this area, and was also very firmly adherent at base on left side under the middle lobe.

The trephining had been over the seat of meningitis. The dura

mater was much detached, particularly over the anterior frontal lobe, and adhered beneath to the gray and white matter.

Membranes showed necrobiotic spots.

Cause of death, pachymeningitis.

Although this dying man's life was not saved by operation, the seat of disease was determined by the symptoms, which was an important matter, since no scalp lesions or accurate clinical history pointed to the exact region of injury. Dr. Mills looks upon the case as one of meningeal disease induced by an injury, causing subcranial extravasation without fracture, which resulted in secondary meningitis, leading to adhesion. It is given here as an example of the value of localizing symptoms.

Enough is shown by these cases to prove the distinct surgical value of the study of cerebral localization, despite the opinion of one of our most distinguished members, who, in 1882, after speaking of the investigations of Broca and Lucas-Championnière, said, "I hope that I may be pardoned for expressing the opinion that their interesting investigations are more ingenious than practically useful."[1]

[1] Ashhurst, Principles and Practice of Surgery, p. 326, note.

CHAPTER II.

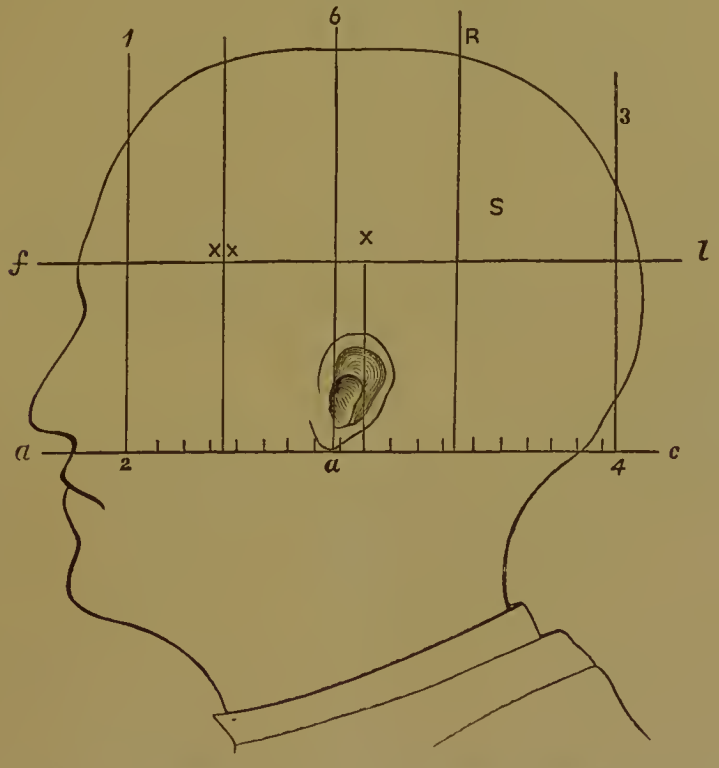
CEREBRAL LOCALIZATION.

I SHALL now devote a little time to the discussion of the means by which the location of the cerebral convolutions important to the surgeon may be marked on the shaven head. The head must be so placed that a line drawn from the alveolar process of the upper jaw, at the base of the incisor teeth to the lowest part of the occipital bone, will be horizontal. Ranney[1] says this line or plane is easiest drawn in the living subject from the cusps of the teeth to the tip of the mastoid process. This alveolo-condyloid line is the base line from which other measurements are made. A perpendicular line drawn upwards from the alveolo-condyloid line through the external auditory meatus corresponds at the top of the skull with the transverse plane running through the bregma or point of intersection of the sagittal and coronal sutures. This line is called the auriculo-bregmatic line. The location of the bregma, which, of course, lies in the median line of the head, is thus determined. Sometimes the bregma, which is the anterior fontanelle of infancy, can be felt through the scalp of adults; if it cannot be so distinguished, the ready methods of Broca or Lucas-Championnière may be employed instead of the mathematical one just described. A draughtsman's square of flexible material, such as steel, copper or tin, which has, at the intersection of the arms, a conical plug to go into the auditory meatus, may be used. The plug is to be placed

[1] Applied Anatomy of the Nervous System, p. 69.

in the ear, the horizontal arm bent until it lies across the upper lip directly under the columella of the nose, and the vertical arm then bent over the top of the head. The bregma is at the point where the axis of this vertical arm crosses the median line of

Fig. 3.



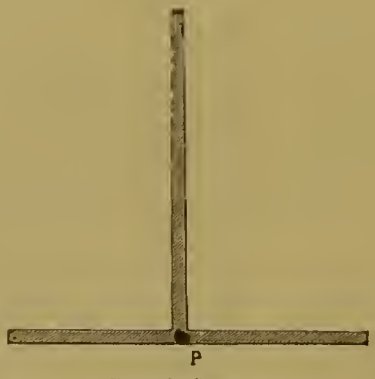
Outline of Head, reduced one-third, on which a modified system of Férè's lines is drawn (Amidon).—*a c*, alveolo-condylold line; *a b*, auricnlo-bregmatic line; *f l*, fronto-lambdoidal line; 1, 2, frontal limit; 3, 4, occipital limit of the brain; *R*, posterior extremity of the fissure of Rolando; *X*, anterior extremity of same; *X X*, location of the middle fold of the third frontal convolution, a little below and in front of which lies the tip of the sphenoidal lobe; *S*, the posterior extremity of the fissure of Sylvius. The intersection of the lines 3, 4, and *f l*, locates the occipto-parietal fissure.

the skull. This probably will give the situation of the bregma more accurately than the original Broca square, which, according to Lucas-Championnière,[1] had the ear plug a little behind the intersection of the arms. The other method of locating the bregma is to cut a section out of a sheet of pasteboard so that it will sit astride of the shaved head from auditory meatus to

[1] Trépanation guidée par les localisations cérébrales, p. 112.

auditory meatus. In order to insure the pasteboard being perpendicular to the alveolo-condyloid line, a lead pencil or stick is thrust at a right angle through the pasteboard on a level with

Fig. 4.



Broca's Square.

the eyes. This enables the surgeon to detect any error in the horizontal position of the base line of the head. The centre of the arch in the cardboard is over the bregma.

Fig. 5.

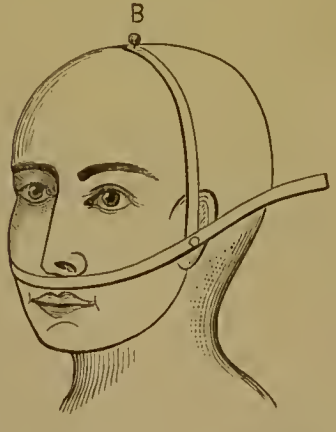


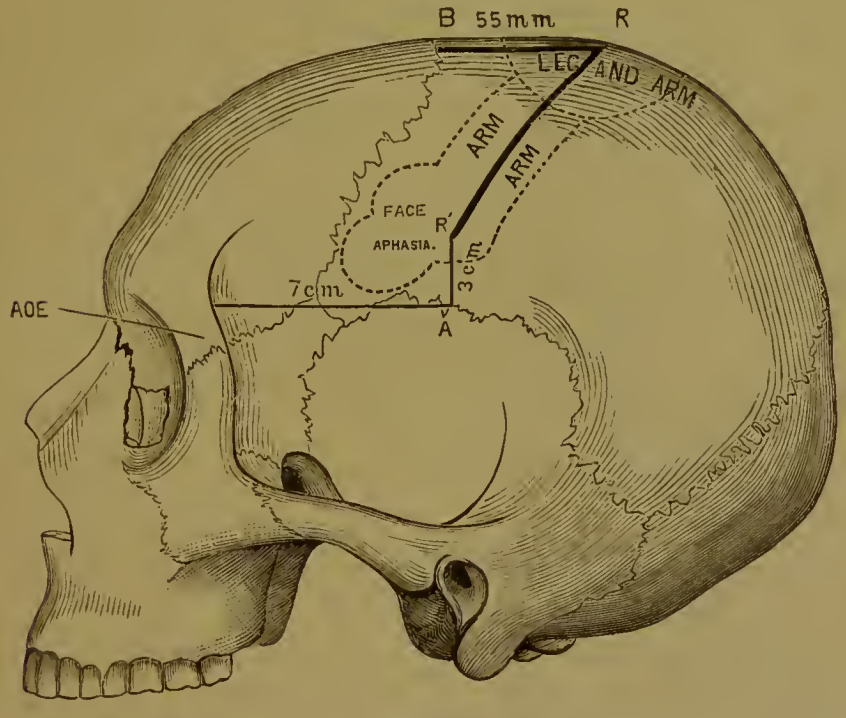
Fig. 6.

Methods of determining bregma.

The most important cerebral sulcus of each cerebral hemisphere concerned in our present studies is the fissure of Rolando, which has its upper end about 5 centimetres behind the bregma, but does not run quite up to the middle line. Some writers give

4.5 centimetres, others 5.5 centimetres. Its lower end lies about 0.5 centimetres behind the auriculo-bregmatic line, and a little above a horizontal line, parallel to the alveolo-condyloid line,

Fig. 7.



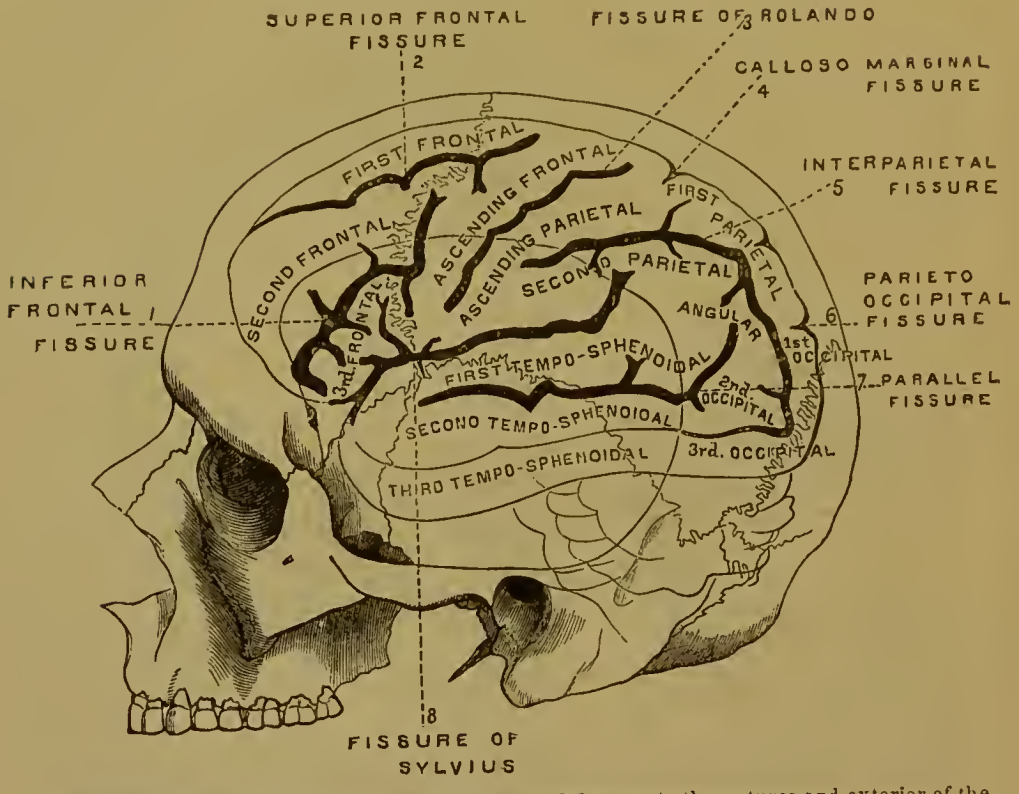
Diagram, from Nancrede, showing the method of locating on exterior of skull the fissure of Rolando, the motor areas of leg, arm, and face, and the centre whose destruction causes aphasia.

[If this is compared with my diagram on page 50 it will be seen that Nancrede gives distance of *R* from *B* as 5.5 mm., instead of 5 c., and marks leg and arm at top of motor area instead of leg alone I prefer the latter arrangement, but give his diagram here in addition to my own, which comes afterwards.—J. B. R.]

carried backwards from the superciliary ridge. This would place the lower end of Rolando's fissure about 6 centimetres above, and a little behind the external auditory canal. Another method of fixing the lower end of the fissure of Rolando is to draw a horizontal line, that is a line parallel to the alveolo-condyloid line, from the external angular process of the frontal bone, where it begins to curve upwards to form the temporal ridge. At a point 7 centimetres distant from the anterior extremity of this line draw a perpendicular line extending upwards 3 centi-

metres. The end of this last line is over the lower end of the fissure of Rolando.

Fig. 8.



Showing relation of the chief convolutions and fissures to the sutures and exterior of the skull (Diagrammatic).

Erichsen quotes[1] Thane as follows: "The upper end of the sulcus of Rolando is placed about half an inch behind a point midway between the root of the nose and the external occipital protuberance; its lower end is close to the posterior limb, and about an inch behind the bifurcation of the fissure of Sylvius. The bifurcation of the fissure of Sylvius corresponds to a point one inch and a quarter behind and a quarter of an inch above the level of the external angular process of the frontal bone."

The occipito-parietal fissure corresponds nearly with the lambdoidal suture at the median line.

1 Science and Art of Surgery, i. p. 731.

After the location of the fissure of Rolando has been marked, as with an aniline pencil, on the scalp, it is easy to locate the cerebral convolutions which control the movements of the opposite half of the head, body, and limbs.

"The first or superior frontal convolution will be found commencing about 2.5 centimetres behind the bregma, and passing forward near the median line toward the orbit. The second frontal convolution will occupy a similar but more lateral position, while the third frontal convolution lies wholly in front of the auriculo-bregmatic line, and on a plane 5 centimetres above the external auditory meatus. Its folded or central part is about 2.5 centimetres in front of the auriculo-bregmatic line, or about 2 centimetres behind the external angular process of the frontal bone."[1]

I have now shown how we can outline upon the scalp the situation of the cerebral convolutions. Hence, if we find symptoms which are known to be produced by lesions of certain regions of the cortex of the brain, it becomes our duty to trephine over the region indicated, provided that the symptoms are sufficiently particularized to indicate that a moderate, I do not say small, area of the brain is affected by disease. Inflammation, abscess, or tumor, while circumscribed, will give a localized group of symptoms; soon, however, secondary œdema, inflammation, or softening may occur; and, by affecting other areas of the brain, will give additional symptoms. These secondary or indirect local symptoms will render accurate delineation of the seat of lesion difficult. Cases, therefore, should be studied accurately and carefully by competent observers at an early period, before the symptoms become more general through advancing disease, and the relief possibly derivable from operation diminished.

Cerebral localization is as valuable when informing us to abstain from operation as when telling that speedy operation is demanded. Loss of motion, or motor paralysis, is the symptom that has been chiefly depended upon as a localizing symptom in surgery, because it is more easily observed, and has been more

[1] Amidon, Medical News, June 21, 1884.

carefully studied then loss of sensation, or sensory paralysis, and the other cerebral functions. The area about the fissure of Rolando is that in which the motor centres for the face and limbs have been found by physiologists and neurologists. It is necessary, therefore, to study these in more detail. As physiological knowledge increases, the sensory area of the brain will, in all probability, become equally important to surgeons.

I have formulated the most important facts according to recent physiological research, in order that the bearing of the corresponding surgical questions may be understood. The progress of knowledge may overthrow some of my conclusions, but that they are quite justified by present knowledge I think undoubted. The physiological portion and the tables which immediately follow, have been submitted to my colleague, Dr. Chas. K. Mills, who, while not responsible for my statements, has kindly given me valuable suggestions. In order to use these facts a general diagnosis of a cortical lesion, that is, of a lesion not of the ganglia or tracts, must first be made.

Motion.—The gray matter of the ascending frontal and ascending parietal convolutions, which are separated from each other by the fissure of Rolando and coalesce over the top of this fissure in the paracentral lobule, is the portion in which are chiefly located the motor centres for the muscles of the face, tongue, arm, and leg of the opposite side of the body, although the motor zone may extend forwards and backwards slightly beyond the strict limits of these convolutions. Some few nerve-fibres are in relation with the muscles of the same side, but nevertheless, in fully 90 per cent. of lesions of the cortical centres the anomalies of motion, namely, spasm and paralysis, occur on the side opposite that on which the brain lesion exists. This statement is subject to modification to be presented when speaking of the rôle of the dura mater. These ascending convolutions are conveniently spoken of as the central region.

The motor area for—

Face and *tongue*	lies in	central	region,	*lower third.*
Arm	"	"	"	*middle* "
Leg	"	"	"	*upper* "

In these areas are the centres for the individual muscles of said limbs, face, and tongue.

Spasm of one of these groups of muscles indicates irritation of the corresponding motor area in the brain cortex.

Paralysis of the same group of muscles indicates destruction of the same motor area.

Spasm or paralysis of two of these groups indicates that the lesion involves more than one area, for example:—

Paralysis of one side of the face and of the corresponding arm indicates a destructive lesion of the lower and middle thirds of the central region.

Paralysis of the arm and corresponding lower limb points to a destructive lesion of the middle and upper thirds of the central region.

Spasm would indicate similarly located irritative lesions.

Paralysis of face, arm, and leg (hemiplegia), would indicate destructive lesion of the lower, middle, and upper thirds of the central region; and spasm of the same muscle, irritative lesion of all three portions of the central region; except that it must be remembered that a primary lesion of one area sometimes affects secondarily the neighboring area or areas and causes hemiplegia.

This is explained by several facts, among which are these—

1. The blood supply to the central region comes entirely from the middle cerebral artery, which ascends in the pia mater upon the surface of the convolutions from the base in the vicinity of the island of Reil. Hence, a lesion in a lower area may cause secondary symptoms in the area or areas above by cutting off its arterial supply.

2. The nerve-fibres converge as they pass inwards from the cortex to make up the bundles traversing the interior of the brain in their journey to the muscles. Hence, a lesion whose influence extends by pressure or otherwise below the surface a short distance may implicate converging fibres, coming from surrounding cortical areas, and thus give results such as would occur if these more distant cortical areas were also really involved.

These and other facts explain the occurrence, in some cases, of hemispasm or hemiplegia, or of extensive spasm or extensive

paralysis, from limited lesions. If the lesion be a small focus of disease, thus giving rise to extensive spasm or paralysis, it usually will first cause symptoms in the group of muscles supplied from the small area primarily affected, or the symptoms in that group will be more marked than in those secondarily involved. This point is of diagnostic importance. Hence the relative time of occurrence of symptoms always demands careful investigation. When monospasm or hemispasm is one of the consensus of symptoms, the seat of the primary lesion may often be determined by study of character of that spasm, especially by attention to the special muscles in which the spasm began. It is likely to be in that part of the cortex which is the area of control for this special group of muscles.

Paralysis of groups of muscles following spasm of the same is characteristic of disease of the central region.

Seguin suggests that a latent hemiplegic state may be discovered, at least in some cases, by an increase of peripheral temperature, as of fingers or toes, on one side, or by the presence of congestion, or erythema, on one buttock.[1] This may be valuable in diagnosticating cerebral hemorrhage from alcoholic coma and similar affections.

Sensation.—Late observation and experiment have proved that disturbance of general sensation also is one of the direct local symptoms of lesions in the central region. These disturbances include the senses of touch, pressure, pain, and temperature, and the sense of location of a limb, and may consist in impairment of these functions or the perception of such sensations when there is no objective cause to produce them (hallucinations of sense). General sensation then is located in the central region, and especially it would seem in the portion behind the fissure of Rolando, that is, the ascending parietal convolution. It must not be forgotten, however, that the sensory region seems to include also the parietal lobes which lie behind the ascending parietal convolution, and which it will be observed contain but few motor centres and fibres. The motor

[1] Gross, System of Surgery, vol. ii. p. 44.

and sensory regions thus seem to overlap; for lesions in front of the fissure of Rolando have caused sensory as well as the usual motor disturbances; and we have seen that motor disturbances may be due to lesion behind the fissure of Rolando in the ascending parietal convolution where I have just said sensory areas exist. It is possible that future and more accurate investigation will show that the motor region is particularly located in front of the fissure of Rolando, but also extends on the posterior side of the fissure, overlapping, or fading into, the sensory region behind the fissure, in a manner similar to that in which the motor areas for the arm and leg appear to overlap or fade into each other. It is more probable, perhaps, that we have not yet sufficiently defined the exact boundaries, because our methods of investigation are not sufficiently accurate. As is the case with the motor centres, which are connected especially with the opposite side of the body, so is it with the sensory centres; though a few sensory fibres go, as do a few motor fibres, to the same side of the body. Hence, sensory lesions give more marked sensory disturbance on the side opposite the lesion, and affect in a less degree the side corresponding to the lesion.

The sensory area for—

Face probably lies in the central region,		*lower third.*
Arm	" " " " "	*middle* "
Leg	" " " " "	*upper* "

The area in each case probably extends also behind the fissure of Rolando in the ascending parietal and parietal convolutions.

Hyperæsthesia, pain, or paræsthesia of the face, arm, or leg, indicates irritation of the corresponding sensory area in the brain cortex.

Anæsthesia and analgesia indicate destruction of the corresponding sensory area in the brain cortex.

Hyperæsthesia or anæsthesia of two cutaneous tracts indicates that the lesion involves more than one area. For example:—

Anæsthesia of one side of the face and of the corresponding arm indicates a destructive lesion of the lower and middle third of the central region, probably situated behind the fissure of Rolando, and also in the parietal convolutions.

Anæsthesia of the arm and corresponding lower limb points to a destructive lesion of the middle and upper third of the central region, probably situated behind the fissure of Rolando, and also in the parietal convolutions.

Hyperæsthesia would indicate similarly located irritative lesions.

Anæsthesia of face, arm, and leg (hemianæsthesia), would indicate destructive lesion of the lower, middle, and upper thirds of the central region, probably situated behind the fissure of Rolando, and in the parietal convolutions; and hyperæsthesia of the same cutaneous tracts irritative lesion of all three portions of the central region, probably situated behind the fissure of Rolando, and in the parietal convolutions.

If motor paralysis is associated with these sensory disturbances in a cortical lesion, the probability is, that we have a lesion involving both the ascending and the parietal convolutions. If the motor paralysis is slight, the lesion probably predominates in the parietal convolutions.

Loss of the Special Senses.—In localizing brain lesions by impairment of the special senses it is important to eliminate all lesions occurring between the cortical centre and the sense organ. For example: loss of sight in brain disease may be due to an increase of intracranial pressure from a tumor causing choked disk; or loss of smell to pressure of a growth on the olfactory nerves after they leave the base of the brain. Such sources of error must be eliminated. This often requires careful study of the case.

Hemianopia, failure to recognize or remember familiar objects, or hallucination of vision, indicates disease probably in the occipital lobes, not far from the angular convolution. If the failure of sight includes the whole field of vision, both hemispheres are involved; if hemianopia only is present, one hemisphere is involved. Blindness of the right half of both eyes, that is, obliteration of the right half of the field of vision, indicates destructive lesion of the left occipital lobe, though it is the left half of the retina which is impaired.

Hemianopia on the left side means lesion of the right side of the brain.

Deafness in one ear or hallucinations of hearing on one side (such as voices and music) indicate disease of the first temporo-sphenoidal convolution of the opposite side. Failure to recognize or remember spoken language indicates lesion of the left first temporo-sphenoidal convolution in right-handed persons and of the right first temporo-sphenoidal convolution in left-handed persons.

Loss or hallucination of smell possibly indicates disease at the base of the brain in the temporo-sphenoidal region, which could not readily be reached by surgical procedures.

Loss of taste has not been sufficiently studied to afford even the suggestive evidence that we possess about disturbances of smell.

Disturbance of other Conscious Mental Actions, such as judgment, reason and self-control, will be exhibited in changed disposition and character of the patient. Such change, if no other general or localizing symptoms exist, indicates disease of the frontal lobes.

Aphasia.—Disturbance in speech power indicates disease in he neighborhood of the fissure of Sylvius on the left side in right-handed persons; on the right side in left-handed persons. It must be recollected that neurologists distinguish three kinds of aphasia.

Aphasia may be due to loss of—

1. Memory of words; whose centre is situated in the first temporo-sphenoidal convolution. (Sensory aphasia.)
2. Memory of voluntary acts necessary to speech; whose centre is situated in the posterior part of third frontal convolution and lower part of central region (ascending frontal convolution). (Motor aphasia.)
3. Connection between the regions just described; which is required to turn thought into speech. This function is located in fibres passing through island of Reil and lower part of central region (ascending frontal convolution). (Aphasia of incoördination.)

There is some difference of opinion between French and German writers as to the distribution of these centres, but for our

surgical purposes the assertions above given are sufficiently accurate. It will be seen that the convolutions mentioned are in close anatomical relation. I have followed on this point and in many other respects the elaborate *résumé* of Dr. Starr, who has analyzed a large number of reported American cases.[1]

Fig. 9.

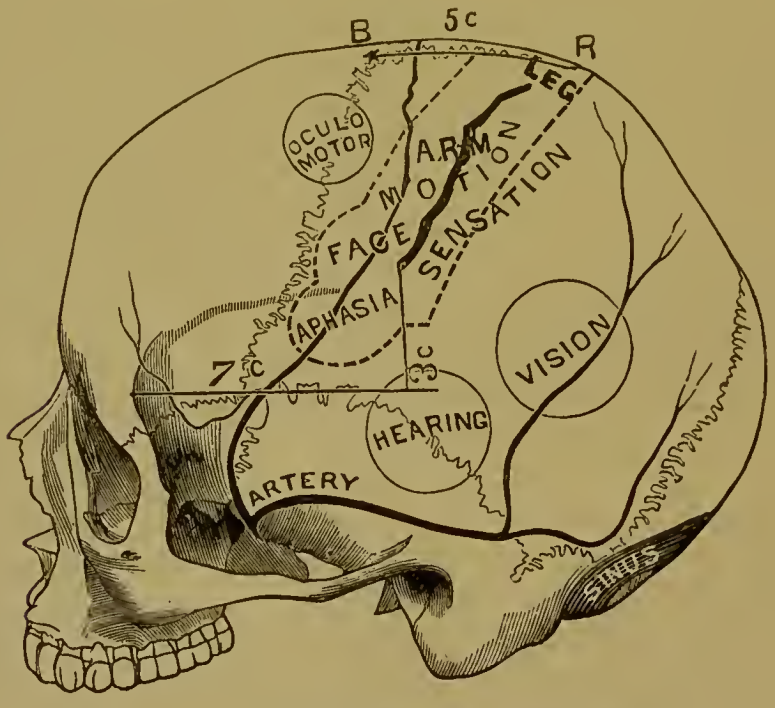


Diagram of the Exterior of the Skull —Upon this have been indicated the measurement for determining the position of the fissure of Rolando; and the location of the regions under which lie the centres of motion and sensation, as well as those of vision and hearing, and the oculo-motor centre. The position of the groovos for the two branches of the middle meningeal artery and for the lateral sinus is also shown.

If the patient understands a question and can recall the words for a reply but cannot make the requisite speech movements he has motor aphasia, which indicates disease in the third frontal and lower end of the ascending frontal convolutions.

If he cannot recognize spoken language, but is able to repeat words after another person, or can use exclamations on being

1 Cortical Lesions of the Brain, by M. Allen Starr, Am. Journ. Med. Sciences, April and July, 1884.

annoyed he has sensory aphasia, which indicates disease in the first temporo-sphenoidal convolution.

If he can understand and talk, but puts an unexpected word for the one desired, he has aphasia of incoördination, which indicates disease situated deeply within the Sylvian fissure or in the island of Reil and the white substance thereabouts, and involving the association fibres.

These points need further study, I admit, because all authorities do not exactly concur in these locations; but the difference is not enough to disturb the question of operation. When opening the skull in the search for abscess or tumor located by localizing symptoms the surgeon should use a large trephine, because the exact spot of lesion is not determinable.

From a study of the points just discussed and the writings of Lucas-Championnière[1] and A. L. Ranney[2] I present the following deductions concerning the proper points for operation, when lesions amenable to operative treatment have been distinguished and localized by the symptoms exhibited. It must be remembered, however, that I am treading very often on debatable ground, and have made statements which from increase in knowledge may require modification in a few weeks or months. I have endeavored to be categorical, and at the same time to indicate by my phraseology those opinions and logical positions which are most satisfactory and certainly proved to be correct.

The researches of H. Duret,[3] who has shown that some motor symptoms may be due to traumatic irritation of the dura mater, must not be forgotten in studying individual cases. The non correspondence of the symptoms, observed in a given case, with the typical symptoms described as accompanying a lesion in that locality, may be due to a dural lesion, in addition to the cortical lesion; and, on the other hand, certain observed symptoms may be caused by dural rather than cortical lesions. These questions are too intricate to be further discussed at this

[1] Trépanation guidée par les localisations cérébrales.

[2] Applied Anatomy of the Nervous System.

[3] On the Role of the Dura Mater and its Nerves in Cerebral Traumatism. See *Brain* (1878), vol. i. p. 29.

time, but must be indicated by me, in order that I may not deceive you into believing that cerebral localization is an easy problem in all cases of brain injury or surgical disease.

Duret says the dura mater contains sensory nerves eminently excitable, and that

"I. Irritative lesions of these nerves cause,

"1. Pain, hyperæsthesia, neuralgia, and reflex motor phenomena.

"2. Reflex spasms or contractures of the muscles of animal and organic life.

"(*a*) The spasms or contractures of the muscles of animal life may occur in the face, eyeballs, neck, trunk, or limbs. They occur sometimes on one side, sometimes on the other." [This is to us an important item.] "These symptoms tend to diffuse and invade neighboring groups of muscles. They have never the localization, the measured and purposed character of the contractions which belong to the lesions of the cortex. They frequently become transformed into permanent contractures.

"(*b*) The reflex vasomotor disturbances, due to irritation of the nerves of the dura mater, consist in spasms or congestive paralysis of the cerebral and ocular vessels, either on the same or the opposite side.

"These facts are important to pathologists, as they show the great influence of irritation of the nerves of the dura mater on cerebral vascular conditions, and on the organs of sense, and on the causation of secondary effects in cerebral traumatism, *i. e.*, on the congestions and inflammations of the cerebral membranes.

"II. Destructive lesions cause local anæsthesia of the dura mater."

I have made this long quotation from Duret because of its importance.

Table I.—Indications in Traumatic Cases.[1]

1. Hemispasm, or 2. Incomplete hemiplegia, or 3. Hemispasm with hemiplegia,	on the opposite side of the body after even slight injury in the fronto-parietal region, and even when not directly over the motor area,	indicates exploratory operation at the site of injury.
4. Monospasm, or 5. Monoplegia, whether total or incomplete,	do. do. do. do.	indicates, indeed demands, exploratory operation at the sit of the injury.
6. Hemihyperæsthesia, or 7. Hemianæsthesia, or 8. Hemianalgesia, or 9. Hemihyperæsthesia with hemianæsthesia or hemianalgesia,	on the opposite side of the body after even slight injury in the parieto-occipital region near or somewhat behind the motor area,	may indicate exploratory operation at the site of injury.
10. Monohyperæsthesia, or 11. Monoanæsthesia, or 12. Monoanalgesia,	do. do. do. do.	may indicate, or may perhaps demand exploratory operation at the site of injury.
The 12 symptoms, above mentioned, if on the same side as the injury, contra-indicate operation at the site of the injury, but		may indicate exploratory operation on the side of the cranium opposite the injury.
13. Coma, with Hemispasm, or Hemiplegia,	in cases of supposed subcranial or arachnoid hemorrhage,	may indicate exploratory operation on the side opposite the paralysis or spasm.
14. Coma, with Hemihyperæsthesia, or Hemianæsthesia, or Hemianalgesia,	in cases of supposed subcranial or arachnoid hemorrhage,	may indicate exploratory operation on the side opposite the hyperæsthesia, anæsthesia, or analgesia.

[1] See note on dura mater and its lesions, p. 52.

Table II.—Contra-indications.

1. Paralysis of one or more cranial nerves, Neuro-retinitis, Cheyne-Stokes respiration, or other symptoms showing lesions probably at the inaccessible base of the brain, or in undeterminable parts of its interior,	even if other symptoms indicate operation are, except in cases of supposed cerebral abscess,	contra indications to exploratory operation.
2. Complete hemiplegia, which often is not of cortical origin, but is due to lesions of deep structures,	is, except when— 1, it is due to a supposed cerebral abscess, 2, it has been incomplete at outset, 3, it is irregular, and corresponds with extensive depression of bone on opposite side of cranium,	a contra-indication to exploratory operation.
3. Hemispasm, or Hemiplegia, with marked hyperæsthesia or anæsthesia, indicates more extensive cortical lesion than hemispasm or hemiplegia alone, and may even be due to deep lesions, and	is, except when— 1, the hyperæsthesia or anæsthesia is limited to a small cutaneous surface (monæsthesia) 2, the hyperæsthesia or anæsthesia is apparently due to the same cortical lesion that causes the spasm or paralysis,	a contra-indication to exploratory operation.
4. Hemispasm, or Hemiplegia on same side as injury,	may, if other symptoms indicate operation, be an indication for operation on the side of the cranium opposite to that of the injury, but	is a contra-indication to exploratory operation on the injured side of the cranium.
5. Hemihyperæsthesia, or Hemianæsthesia, or Hemianalgesia, with marked spasm or paralysis, indicates more extensive cortical lesion than hemihyperæsthesia or hemianæsthesia, hemianalgesia alone, and may even be due to deep lesions, and	is, except when— 1, the spasm or paralysis is limited to a small group of muscles (monoplegia), 2, the spasm or paralysis is apparently due to the same cortical lesion that causes the hyperæsthesia or anæsthesia,	a contra-indication to exploratory operation.
6. Hemihyperæsthesia, or Hemianæsthesia, or Hemianalgesia, on same side as injury,	may, if other symptoms indicate operation, be an indication for operation on the side of the cranium opposite to that of the injury, but	is a contra indication to exploratory operation on the injured side of the cranium.

Note.—When the symptoms indicate that many centres are involved, and this is usually the case, more bone must be removed than by a single trephine perforation. The burr of the surgical engine, or the gnawing bone forceps, is the most convenient instrument for enlarging the trephine hole.

TABLE III.—POINTS FOR OPENING THE CRANIUM WHEN OPERATION IS INDICATED.

Symptoms.	Probable area of cortex involved.	Point where brain should be uncovered.	Remarks.
Hemispasm or hemiplegia.	Along entire length of fissure of Rolando, or possibly along a portion of its extent, and involving other areas secondarily, more probably in front rather than behind fissure.	Over middle of fissure of Rolando, and to be extended downwards, upwards, and forwards in search for lesions, rather than backwards, unless disturbances of sensation have been present; then backward extension is indicated. If aphasia is present the opening should be extended downwards and forwards.	
Crural monospasm or monoplegia (convulsions or paralysis limited to one leg, or part of one leg).	Upper third of central region in ascending convolutions and astride of upper end of fissure of Rolando. Possibly in front rather than behind fissure (?). Some say in ascending parietal convolution rather than in frontal (see Erichsen, i. p. 725, and Lucas-Championnière).	Over upper end of fissure of Rolando, and to be extended forwards rather than backwards, unless disturbance of sensation is present.	
Spasm or paralysis of both legs; not due to spinal lesion.	Upper part of central region, and paracentral lobules on both sides.	Over middle line between upper ends of both fissures of Rolando, to be extended towards the side of vault of cranium which is opposite to the side of the more pronounced symptoms. Take care to avoid if possible injury to superior longitudinal sinus.	
Brachial monospasm, monoplegia (convulsions or paralysis limited to one arm, or part of one arm).	Middle third of central region in ascending convolutions, especially in ascending frontal.	Over middle third of fissure of Rolando, but rather more in front than directly over the fissure, though the fissure should be uncovered. To be extended forwards rather than backwards, unless disturbance of sensation is present.	Facial spasm or paralysis is often associated with the crural symptoms; as by hemorrhage gravitating downwards, etc.
Associated crural and brachial monospasm or monoplegia.	Upper two-thirds or at junction of upper and middle thirds of central region, probably in ascending frontal rather than ascending parietal convolution.	Over junction of upper and middle thirds of fissure of Rolando.	

TABLE III.—*Continued.*

Symptoms.	Probable area of cortex involved.	Point where brain should be uncovered.	Remarks.
Facial spasm or paralysis.	Lower third of central region, especially in ascending frontal and posterior end of second frontal convolutions. The anterior part of this area seems to control chiefly the motions of the upper part of the face, and the posterior part the motions of the lips or mouth.	In front of lower third of fissure of Rolando, though the posterior edge of opening should be close to the line of the fissure of Rolando.	Aphasia is often associated with the facial symptoms.
Aphasia.	Below and in front of lower end of fissure of Rolando, varying somewhat with form of aphasia. 1. Sensory aphasia in first temporo-sphenoidal convolution. 2. Motor aphasia in posterior part of third frontal and lower part of ascending frontal convolutions. 3. Aphasia of incoördination in vicinity of island of Reil. Aphasia in right-handed persons is due to lesion on left side of brain; in left-handed persons to lesion on right side of brain.	About a half inch (1.25 centimetres) below, and a half inch (1.25 centimetres) in front of the lower end of the fissure of Rolando.	Single large opening will cover the convolutions for the three forms of aphasia, but the area for the third form is deeply situated in the fissure of Sylvius.
Associated brachial and facial spasm or paralysis.	Convolutions involved are those of arm and face as given above.	In front of junction of middle and lower thirds of fissure of Rolando.	
Associated brachial and facial spasm or paralysis with aphasia.	Convolutions involved are those of arm, face, and speech as given above.	In front of lower third of fissure of Rolando, and to be extended upwards or downwards and forwards according as brachial or aphasic symptoms are more pronounced.	
Associated facial spasm, or paralysis and aphasia.	Convolutions involved are those of face and speech as given above.	About a half inch (1.25 centimetres) directly in front of the lower end of the fissure of Rolando, and to be extended upwards and backwards, or downwards and forwards, according as facial or aphasic symptoms are more pronounced.	

Table IV.—More Uncertain Localizations.

Symptoms.	Probable area of cortex involved.	Point where brain should be uncovered.
Hemihyperæsthesia, Hemianæsthesia, Hemianalgesia.	Behind fissure of Rolando in ascending parietal and first and second parietal convolutions.	Behind the middle of fissure of Rolando, and to be extended backwards rather than forwards.
Crural monohyperæsthesia, Crural monoanæsthesia, Crural monoanalgesia.	Upper third of ascending parietal convolution and portion of parietal convolutions just behind.	Behind upper third of fissure of Rolando.
Hyperæsthesia of both legs, Anæsthesia of both legs, Analgesia of both legs. Not due to spinal lesion.	Same region as case above, but on both sides of the brain.	Over middle line behind the upper ends of both fissures of Rolando, to be extended towards the side of the vault of cranium opposite to the more pronounced symptoms. Take care to avoid if possible injury to superior longitudinal sinus.
Brachial monohyperæsthesia, Brachial monoanæsthesia, Brachial monoanalgesia.	In middle third of ascending parietal convolution and portion of parietal convolutions just behind.	Behind middle third of fissure of Rolando.
Associated crural and brachial Monohyperæsthesia, Monoanæsthesia, Monoanalgesia.	Upper two-thirds of ascending parietal convolution and portion of parietal convolutions just behind.	Behind middle two-thirds of fissure of Rolando.
Facial hyperæsthesia, Facial anæsthesia, Facial analgesia.	Lower third of ascending parietal convolution and portion of parietal convolutions just behind.	Behind lower third of fissure of Rolando.
Associated brachial and facial Monohyperæsthesia, Monoanæsthesia, Monoanalgesia.	Lower two-third of ascending parietal convolution and portion of parietal convolutions just behind.	Behind lower two-thirds of fissure of Rolando.

TABLE IV.—*Continued.*

Symptoms.	Probable area of cortex involved.	Point where brain should be uncovered.
Loss or change in power of attention, reason, judgment, or self-control.	Frontal lobes.	Surface of frontal bone, but the spot could only be located by some other symptoms which might be present, such as aphasia ocular deviation, affections of the leg or arm.
Muscular deviations of the eye and dilatation of the pupil.	In first and part of the second frontal convolutions in front of motor centre for arm, reaching nearly to the longitudinal fissure.	About three centimetres in front of spot for exposing motor centre of arm.
Blindness or failure to recognize and remember familiar objects, or hallucinations of vision.	Occipital lobes near parietal lobes, not far from angular convolution. Blindness of half field of both eyes at *right* side of body indicates destruction of *left* lobe, and vice versa.	About seven centimetres above the external occipital protuberance, and seven centimetres around the skull laterally.
Deafness, or hallucinations of hearing.	First temporo-sphenoid convolution of side opposite to affected ear. Failure to recognize or remember *spoken* language is a form of aphasia (sensory aphasia), and is due to lesion in first temporo-sphenoidal convolution.	Just below and about three centimetres behind the lower end of the fissure of Rolando.
Loss or hallucinations of smell.	Probably at base of brain in temporo-sphenoidal region.	Not accessible to operation.
Loss or hallucinations of taste.	Not located.	

CHAPTER III.

OPERATIVE TREATMENT OF CEREBRAL LESIONS.

I HAVE now with great detail discussed what I conceive to be the principles of the operative surgery of the human brain. It remains to apply these principles to the treatment of—

A. Cranial fractures.
B. Intracranial hemorrhage.
C. Intracranial suppuration.
D. Epilepsy following cranial injury.
E. Insanity following cranial injury.
F. Cerebral tumor.

A. In discussing the treatment of cranial fractures I shall ask myself four questions:—

1. What conditions demand incision of the scalp?
2. What conditions render incision of the scalp unjustifiable?
3. What conditions demand perforation of the skull?
4. What conditions render perforation of the skull unjustifiable?

These queries are best answered by the tabulated statement which follows. I admit that the line of treatment advocated is more heroic than that generally taught, but it has been accepted only after careful consideration of the reasoning of those who hold the opinion contrary to my own. Every case must be individually studied, and the patient's chances of death, of life, with subsequent epilepsy or insanity, or of return to perfect health, carefully weighed; but for a working rule to guide the student and

practitioner I think experience will show that the indications given in the table are correct. Trephining, properly performed, is in itself so free from danger that in a doubtful case the patient had better be trephined than allowed to run the risk of death, epilepsy, or insanity. Legouest was very nearly right when he said: "Whenever there is a doubt whether trephining should be done, this doubt is probably an indication that operation should be performed."[1]

Syllabus of the Treatment of Fractures of the Cranium.

CLOSED FISSURED FRACTURES.

1. No evident depression, no brain symptoms. No operation.
2. No evident depression, with brain symptoms. Incise scalp and trephine.
3. With evident depression, no brain symptoms. Incise scalp and possibly trephine. (See note below.)
4. With evident depression, with brain symptoms. Incise scalp and trephine.

CLOSED COMMINUTED FRACTURES.

5. No evident depression, no brain symptoms. Incise scalp and probably trephine. (See note below.)
6. No evident depression, with brain symptoms. Incise scalp and trephine.
7. With evident depression, no brain symptoms. Incise scalp and trephine.
8. With evident depression, with brain symptoms. Incise scalp and trephine.

OPEN FISSURED FRACTURES.

9. No evident depression, no brain symptoms. No operation, but treat wound.
10. No evident depression, with brain symptoms. Trephine.
11. With evident depression, no brain symptoms. Possibly trephine. (See note below.)
12. With evident depression, with brain symptoms. Trephine.

[1] Lucas-Championnière, Trépanation guidée per les localisations cérébrales, p. 23.

OPEN COMMINUTED FRACTURES.

13. No evident depression, no brain symptoms. Probably trephine. (See note below.)
14. No evident depression, with brain symptoms. Trephine.
15. With evident depression, no brain symptoms. Trephine.
16. With evident depression, with brain symptoms. Trephine.

PUNCTURED AND GUNSHOT FRACTURES.

17. In all cases and under all circumstances. Trephine.

NOTE.—In classes 3 and 11 I should be inclined to trephine if the depression was marked, or the fissures sufficiently multiple to approach the character of a comminuted fracture. In classes 5 and 13 I should trephine, unless the comminution was found to be inconsiderable.

The operation, when decided upon, should be performed at once, or certainly not delayed more than a few hours.

All cases, whether trephined or not, should be treated as cases of incipient inflammation of the brain.

B. In deciding upon operative treatment of intracranial hemorrhage, two questions are to be answered:—

What symptoms render perforation of the skull, in order to remove the clot and secure the bleeding vessels, unjustifiable?

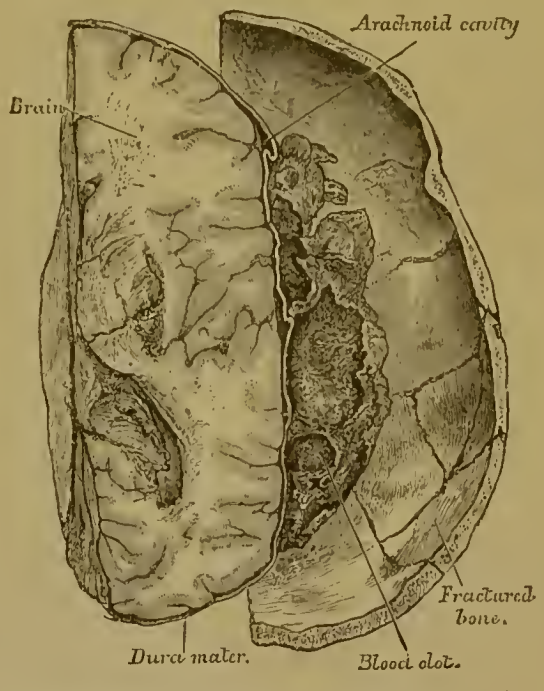
What symptoms demand such operation and render it justifiable?

Intracranial hemorrhages occur between the cranium and dura mater, in the cavity of the arachnoid, in the meshes of the pia mater, in the substance of the brain and in the ventricles. Hemorrhages within the ventricles or in the substance of the brain, whether traumatic or idiopathic, must with our present knowledge be treated solely by medical means; though I consider it not impossible that medical science may in time teach us to locate and diagnosticate such lesions with a precision that may justify exploratory and therapeutic operations in a limited number of cases. Blood poured out in the meshes of the pia mater is usually associated with laceration of the brain, and spreads so widely over the surface of the convolutions, that it is impossible to locate the origin of the bleeding or to remove the distributed

blood. Hence, operation is contra-indicated in cases of intracranial bleeding that do not present the symptoms which are believed to be produced by accumulation of blood in either the cavity of the arachnoid or the space between the skull and dura mater.

The differential diagnosis between circumscribed hemorrhage within the arachnoid cavity and circumscribed hemorrhage between the dura and cranium is practically impossible, though it is said that marked inequality of the pupils and marked hemiplegia are less noticeable in arachnoid extravasation than in sub-

Fig. 10.



Hemorrhage from Meningeal Artery. (BRYANT.)

cranial extravasation. If localized hemorrhage within the arachnoid is reasonably ascertained, trephining with incision of the dura is in my opinion justifiable. After the bone is removed the bluish and non-pulsatile dura will probably bulge into the opening, and should be incised to allow escape of the blood. Nancrede refers[1] to cases where the bulging of the dura did not

[1] International Encyclopædia of Surgery, vol. v. p. 51.

occur until many hours had elapsed. It is not likely that any special vessel will require ligation or similar treatment in arachnoid hemorrhage; as occurs not infrequently in subcranial hemorrhage, where the bleeding often comes from a large meningeal artery or a venous sinus.

Hemorrhage between the dura and cranium (subcranial extravasation) is not very unusual as an accompaniment of head injuries, because the branches of the large middle meningeal artery ramify over the inner surface of the parietal bone, which, from its position, so often bears the brunt of the vulnerating force. Indeed, the artery is sometimes, especially at the lower anterior angle of the bone, enclosed in a bony canal, so that fracture of the bone at that point almost insures the laceration of the artery. The venous sinuses are also torn or wounded at times, and cause subcranial extravasation. Hemiplegia on the side opposite the extravasation, rapidly followed by general paralysis and coma, is frequently seen in subcranial hemorrhage. Convulsions during the time of bleeding occur, though not very frequently.

In bleeding from the middle meningeal artery, the clot often causes pressure at the base of the skull, and therefore may, by pressure on the cavernous sinus and nerves going to the eye, cause on the side of injury protrusion of the ball and dilatation of the pupil, or wider dilatation than in the other eye; while the hemiplegia from cortical pressure will be on the opposite side. If a fissure exists the blood may escape into the temporal fossa of the injured side and cause tumefaction there. Erichsen records two cases where these symptoms were a valuable aid in diagnosis.[1] Such symptoms, as well as embarrassment of respiration, indicate operation; when the blood lies within the brain, because due to cerebral laceration, the patient has, if the laceration is at all severe, no interval of consciousness after the injury; concussion symptoms soon give place to compression symptoms; and paralysis is not complete, as a rule, and is apt to be limited to hemiplegia. Twitching of the limbs, convulsions of the body, restlessness, and muttering incoherence, contracted or irregularly

[1] Surgery, vol. i. p. 735.

dilated and contracted pupils with perhaps squinting, are the prominent symptoms which occur.

The diagnosis of traumatic subcranial hemorrhage, and indeed, in a surgical sense, the same may be said of arachnoid hemorrhage also, centres upon the absence of the characteristic symptoms of laceration just given, and upon the fact that the group of symptoms commonly called "compression of the brain" occur not immediately after the injury, as in laceration of the brain or hemorrhage into the ventricles or brain substance, but later, that is, after the interval of a few minutes or hours. For example, a man is struck on the head and falls stunned, but soon recovers consciousness, though subsequently he becomes comatose and paralyzed on the side opposite the injury. Here the presumption of a subcranial or arachnoid hemorrhage having occurred, as soon as reaction has sent enough blood to the head to distend the torn vessel, is very great. This period of immunity from head symptoms after traumatism is sufficient to justify operative search, when an injury or such symptoms as coma, dilated pupil, hemiplegia, monoplegia, or monospasm, sufficiently localize the effusion; or if the symptoms are increasing or are of sufficient severity to point to the probability of active encephalitis. Apoplexy, drunkenness, opium poisoning, uræmic coma and intracranial hemorrhage are often to be differentiated in cases without history. The problem is frequently difficult. Reference has been made, on page 46, to Dr. Seguin's suggestion regarding the diagnosis of latent apoplectic paralysis. A very short interval indicates rapidly flowing hemorrhage and demands early operation, with probably ligation of the middle meningeal artery or one of its large branches, or ligation or compression of a sinus. It must be remembered also that compression symptoms, coming on immediately or quite soon after depressed cranial fracture, are very often due to the subcranial clot rather than the depressed bone. Removal or elevation of bone is often needed more for liberation of blood-clot and control of hemorrhage than for deformity in the bony outline.

Rapid hemorrhage will probably be from the middle meningeal artery, and therefore a large trephine should be put over

the anterior inferior angle of the parietal bone, and the squamous portion of the temporal bone just below this angle, unless the localizing symptoms direct to some other point. Inspection of the skull will show that the two large branches of this artery, as they ascend to distribute branches to the lateral region of the cranium, can both be uncovered by a large trephine applied at this point. This point is situated about 1½ inches behind the external angular process of the frontal bone and 1 inch above the zygoma, or 1½ inches directly above the condyle of the jaw.

The rules given by Keen and Erichsen are accurate,[1] I think, for uncovering the superior branch, but place the trephine a little too high, if the two branches are to be uncovered at their point of divergence. There are probably variations of the point of bifurcation. The bleeding vessel when found may be ligated or twisted, or the bony canal plugged with a piece of stick or wax, or the actual cautery applied by means of a red hot needle. Nancrede[2] has collected 40 cases of operation of this kind with 24 recoveries and 16 deaths. Early operations gave, as might be expected, a much better percentage of recovery than those in which operation was delayed.

The tendency has been to deter surgeons from operating in intracranial extravasations, unless the certainty of diagnosis was great. For example, Gross says:[3] "The only case in which such a procedure is really warrantable, is where the extravasation is associated with, or dependent upon, fracture of the skull, complicated with depression, or serious injury of the soft parts, or where the fracture is situated directly over the course of the middle meningeal artery." This advice is, in my opinion, too restrictive. Gradually increasing brain symptoms after head injury, especially if there has been a period of return to consciousness after shock of traumatism, suggest the occurrence of intracranial bleeding from meningeal vessels, even when no fracture or depression of bone is detectable. Operation is then especially

[1] Holden's Medical and Surgical Landmarks. Edited by W. W. Keen, Phila., 1881, p. 18. Erichsen's Science and Art of Surgery, i. p. 739. See Fig. 9, p. 50.

[2] International Encyclopædia of Surgery, vol. v. p. 48.

[3] System of Surgery, vol. ii. p. 55, ed. 1882.

indicated, if the localizing symptoms point to no other region of the cortex and if symptoms such as are described on p. 62 do not render cerebral laceration probable.

C. I come now to the operative treatment of intracranial suppuration.

I believe that operative evacuation should be undertaken in all cases of localized intracranial suppuration, whether the abscess is subcranial, intrameningeal, or cerebral. Ashhurst[1] and some other authorities rather deprecate operation, because patients have lived a long time with cerebral abscess in a quiescent, encysted state; and, perhaps, also, because autopsies have in rare instances shown what appear to be abscesses of the brain cured by caseation. The tendency, however, of intracranial suppuration, whether diffused or localized, is known to be towards death. Cerebral œdema, progressive invasion of vital centres, rupture of the abscess into the ventricles, and extension to the surface of the base where fatal meningitis occurs, are the usual modes of destroying life. Spontaneous evacuation through the nares, orbit, ear, or bony wall is too rare to be expected in any given case. Absorption, as far as we know, never occurs.

The difficulty in every case is that of establishing a diagnosis of pus, and proving that the pus, if present, is not a diffused meningeal exudation on the surface of the brain. The occurrence of symptoms indicative of intracranial suppuration, within a few days of the receipt of a cranial injury, usually means diffuse meningeal suppuration and contra-indicates efforts at evacuation. Symptoms of intracranial suppuration occurring at a later period are more apt to be due to abscess in the cerebral tissue, or to a localized pus collection between the bone and the membranes, and therefore indicate more decidedly the propriety of operation with a view to evacuation.

Intracranial suppuration, such as may be benefited by operation, provided that the exact situation of the pus can be diagnosticated, is apt to present a combination of some of the following symptoms:—

[1] Principles and Practice of Surgery, p. 326.

1. Headache, or a subjective sense of pressure on the brain. The headache is, perhaps, usually dull, and when present is quite constant.

2. Giddiness, nausea, or even vomiting.

3. Mental hebetude, dulness of senses, slowness in speech and movements, aphasia, mental irritability, muttering delirium, increasing imbecility.

4. Constipation.

5. Paresis, sensory abnormalities.

6. Sleeplessness, optical delusions, attacks of terror.

7. Chills.

8. General convulsive seizures of an epileptiform character; sometimes in a few cases localized convulsions.

9. Little, if any, elevation of general bodily temperature.

10. Perhaps localized increase of surface temperature of the head.[1]

At a later period there will occur most probably—

1. Hemiplegia.

2. Perhaps active delirium, severe headache, high temperature, especially if meninges become involved secondarily.

3. Coma.

4. Passive retinal congestion.

5. Involuntary evacuations.

6. Slow and full pulse, dilated or sluggish pupils.

7. Respiratory failure.

8. Death.

The symptoms of chronic abscess may be so insidious as to escape notice of all except a very careful observer. It has been said that there are at times no symptoms. This I doubt, believing that a critical examination will detect them, especially if the observer be trained in investigating nervous diseases and be sufficiently intimate with the patient to have known his previous habits and mental characteristics.

Puffy swelling of the scalp, and separation of the pericranium,

[1] The suggestion of Flitner to measure the temperature within the external auditory meatuses by a properly constructed thermometer might be utilized. See Journal of American Medical Association, Nov. 15, 1884.

with dry and yellow bone beneath it seen at the bottom of a head wound, will in some few cases be found, and with general symptoms of pus, become a clear evidence of abscess beneath the bone at that spot.

Symptoms, such as just mentioned, demand trephining. If the pus is not found beneath the bone, and the dura is pulseless and bulging, that membrane should be incised. If no pus is discovered, and the general symptoms of intracranial abscess are marked, exploratory puncture and aspiration of the brain should then be performed. In some cases, the surface thermometer may indicate the point on the cranium nearest the focus of suppurative encephalitis.

Nancrede[1] believes that when an intracranial abscess involves cerebral tissue alone the general bodily temperature is subnormal, or at least is not an elevated temperature. He looks upon high temperature as an evidence of localized suppurative arachnitis, or a meningitis in addition to the cerebral abscess. Cerebral abscess secondary to bone disease would, of course, be liable to present an elevation of temperature, because meningitis was an early concomitant.

If a majority of the symptoms tabulated as occurring in intracranial abscess exist and there has been received an injury on the side of the head opposite the hemiplegia, to which the supposed abscess can, with reason, be attributed, an exploratory operation should be done at the point of injury.

If the said symptoms exist and the principles of cerebral localization indicate any special region of the cortex or brain substance, an exploratory operation should be done on that part of the skull that affords best access to the supposed seat of lesion.

Incision of the membranes, and exploratory puncture of the brain substance, with aspirating tubes, to the depth of three or more inches in various directions is justifiable if the symptoms of abscess are undoubted. The abscess cavity or cavities when found should be drained by free openings and counter-openings, and drainage-tubes; and should even be washed out by hydrostatic

[1] International Encyclopædia of Surgery, vol. v. p. 86.

irrigation, if it is probable that there are pockets in which pus may remain and decompose.

Scarcely any limitation should be dogmatically given regarding the operative search for pus in undoubted abscess of the cerebral tissue, so long as the aspirating tube is not thrust deeply towards the vital centres located in the medulla oblongata and base of the brain. It is never too late to seek for the pus, for Nancrede gave a patient several days of life after he was thought, by several competent observers, to be dead, by promptly opening by incision a cerebral abscess. The earlier the operation is performed the better; but it is never too late to make the attempt at evacuating an intracranial abscess.

D. In epilepsy following injury much good may at times be effected by the removal of the cranial wall at the site of traumatism.

An internal spicule, or osteophyte from depressed or chronically inflamed bone, penetrating or pressing upon the membranes, thickened bone, pathological changes in the membranes themselves, the existence of a sequestrum of necrotic bone, depressed and neuralgic cicatrices of the soft parts and similar kinds of irritation, may be the exciting cause giving rise to epilepsy in patients whose convulsive centres are especially irritable. Briggs has seen cases of epilepsy in which narrow spurs of bone nearly a half inch long grew from the inner surface of the skull.[1] Removal of the cause by operation is in such case certainly demanded. Epilepsy due to peripheral irritation of a cicatrix in the scalp without bone lesion may be relieved by dissecting out the scar tissue or detaching it as a flap from the bone and allowing healing by granulation to occur. The application of the actual cautery to the scar may, perhaps, destroy the focus of irritation; though in a case where I tried this method no relief was obtained.[2]

Cauterization or excision of scar tissue should be attempted in traumatic epilepsy therefore before resort is had to trephining,

[1] See Duret's paper on the Dura Mater, to which reference is made on p. 52.

[2] See TRANSACTIONS OF THE AMERICAN SURGICAL ASSOCIATION, 1884, p. 132.

except, perhaps, in cases where there is an evident accentuated depression of the bone. Then the irritating cause of the convulsions is not likely to be in the soft tissues alone.

The subject of trephining in epilepsy was so ably and thoroughly handled at the last meeting of the Association by our present President, Dr. Briggs,[1] that I shall do little more than repeat some of his statements and add a few thoughts which may serve to impress the Fellows with the importance of the topic and cause them to again read his valuable paper.

The development of epileptic convulsions requires, first, a persistent abnormal excitability of the centres in the medulla oblongata and pons Varolii; and, secondly, a peripheral irritant. The latter is the exciting cause, and may be hard to discover in any given case. Traumatism often furnishes the peripheral irritant in the existence of a sensitive scar in some part of the head or body, or a cranial lesion irritating the cerebral membranes or brain. The hyper-excitability of the medulla and pons may be inherited, or acquired through personal accidental causes. Echeverria attributes[2] ten per cent. of all epilepsies to injuries of the head; but it must be remembered that the convulsions may not be developed for years after the injury. Briggs has seen cases in which ten years had elapsed, and he quotes a case of Dudley's which developed sixteen years after injury, and was entirely cured by trephining. I must admit, however, that Althaus has stated that fourteen years' experience in a hospital devoted to epilepsy has taught him that traumatic epilepsy is rare.[3]

If operative treatment is instituted for traumatic epilepsy, all the diseased or depressed bone should be removed. Briggs has taken away the cranial wall covering an area as large as the palm of the hand, and cured the epilepsy without causing an unpleasant symptom.

From tables of the operation collated by Stephen Smith, Billings, and Echeverria, 92 American operations are reported, of which 63 were cured, 13 ameliorated, 2 not changed, 14 died.[4]

[1] Transactions of the American Surgical Association, 1884, p. 132.

[2] Briggs, Ibid.

[3] Amer. Jour. Med. Sciences, January, 1880, p. 270.

[4] Quoted from Briggs.

Walsham has collected 82 cases, to which he has added 48 collected by Billings and others, making 130 in all, of which number 75 were completely cured, 18 improved, 7 unimproved or worse, 30 died.[1] 30 cases of epilepsy from old injuries of the head have been operated upon by Briggs. Of these, 25 were cured, 3 ameliorated, 1 not changed, 1 died.

In 48 of 82 cases, collected by Walsham[2] himself, cure was effected, 13 were relieved at the time of report, 4 were not improved, and 17 died; which gives a death-ratio of 20.7+ per cent., but some of the deaths occurred so long after the operation as to be scarcely attributed with justice to the operation.

Nancrede says[3] that after adding 37 cases collected by himself to the whole number collected by Walsham (130), he finds in the 167 cases a mortality of 19.16 per cent., but does not describe the kind of cases in which death occurred. Cure has in a number of cases followed operation, though no pathological change was recognized in bone or membranes.

In epilepsy and insanity from traumatic causes, connected with the skull, the lesions appear, according to Walsham, to have generally existed in the anterior half segment of the cranium.[4]

The symptoms justifying operation are cicatrices which are sensitive, tender, or abnormally hot by the surface thermometer; pain or sense of brain pressure at the seat of old injury, which may not be constant but which is always, when present, referred to the same spot; pain, vertigo, or convulsions produced by pressure upon the cicatrix; depressions or elevations in the bone; fistules leading to necrotic bone; and any symptoms indicating through study of cerebral localization brain lesions in the region of the external scar. Operation seems to me to be especially indicated in these cases if the scar is in front of the middle of the cranium.

The symptoms contra-indicating operation on the head are

[1] St. Bartholomew's Hosp. Reports, 1883, p. 130.

[2] Ibid., pp. 130 and 136.

[3] International Encyclopædia of Surgery, vol. v. p. 101.

[4] On Trephining the Skull in Traumatic Epilepsy, St. Bartholomew's Hospital Reports, 1883, p. 139.

the existence of other foci of irritation, as in one of Briggs's patients who had a depression of the skull and diseased bone in one leg, and in whom removal of the necrotic tissue in the leg cured the epilepsy without any surgical interference with the head. The absence of pain, tenderness, or other symptom of irritation, the history of epilepsy in other members of the family, or a short period of medicinal treatment would be facts that should cause hesitation in operation until every other means has been employed, or until the progressive character or the severity of the epilepsy forebodes early impairment of intellectual functions.

If operation fails to relieve, it may be repeated near the old spot of operation after the lapse of several months; for at least one case is known where operation was ineffectual, and subsequent autopsy showed a fragment of bone pressing upon the encephalon. Removal of this would probably have cured the epilepsy. Several months' time must elapse before the operation is declared unsuccessful; for the brain does not always give up its convulsive habit at once. Subsequent treatment to relieve the hyper-excitability of the medulla and pons and to remove any other causes of peripheral irritation must be judiciously carried out. The cicatrix left after trephining may become a secondary peripheral irritant and need excision to relieve pinched nerve filaments. I know, however, of no such case on record.

How long should operation be delayed in epilepsy presumably caused by a depressed fracture of the skull or other localized traumatic irritation? Certainly not at all after the diagnosis of the cause has been made, provided that medical means have been efficiently tried and have failed. Operations done promptly prevent an increased development of abnormal excitability in the convulsive centres, and give, therefore, the best chance of recovery from the epileptic manifestations. Mental deterioration increases with the lapse of time, as does, in my opinion, the risk of serious cerebral reaction after operation. A normal nervous system would be less likely to assume inflammatory symptoms dangerous to life than would a nervous tissue long subjected to irritating influences. If the epilepsy cannot be definitely at-

tributed to the traumatism which by the history and by the scar is known to have occurred, I should advocate an empirical operation in the course of five or six months or even earlier; provided that no other cause is discovered, that medicinal treatment has proved unavailing, and that the epilepsy is increasing in severity.

E. Operations for the relief of insanity due to cranial traumatism have not been very generally advocated or attempted. One year ago, Dr. William A. Byrd brought the subject to the attention of this Association,[1] and referred to four cases which were trephined for insanity after fractured skull. Three were operated upon by Dr. W. T. Briggs; the fourth by Dr. Byrd himself. Dr. Byrd's case improved for a short time and then became progressively worse in regard to her mental condition. Two of Dr. Briggs's cases entirely recovered their mental health; one was a boy; the other a man, who had received a gunshot fracture above the ear. The third of Briggs's cases was an almost hopeless one, which he had at one time declined to subject to operation. The lamentable condition of the imbecile patient and the solicitations of the family finally induced him to operate. Death occurred on the second day, after "a rigor followed by great depression."

In the discussion following Dr. Byrd's paper, Dr. T. F. Prewitt is reported[2] as having spoken of trephining an insane woman, who was relieved only for a time. The report here is incorrect, for Dr. Prewitt has written me that he has never trephined for insanity. The remarks, I think, should be ascribed to Dr. Byrd, and refer to his case which formed the subject of debate.

I find a case of insanity cured apparently by operation, mentioned by Dr. L. A. Stimson, and another by Dr. J. L. Little.[3] In his elaborate table of trephining cases, Amidon[4] refers to

[1] Transactions of the American Surgical Association, Vol. II., 1884, p. 130.

[2] Ibid., p. 134.

[3] Annals of Anatomy and Surgery, 1883, viii. p. 137.

[4] Annals of Surgery, March, 1885, p. 211, quoted from American Practitioner, 1883, xxvii. p. 237.

McCormick's case of acute mania, occurring eight years after a depressed cranial fracture, which recovered after the depressed area of bone was removed by operation. In this instance there was pain at the site of wound, which was situated at the junction of the sagittal and coronal sutures.

With so few cases as are reported it is difficult to formulate dogmatic rules. It seems to me, however, that the operation of removing a portion of the cranium is justifiable when, within a few months after a depressed fracture of the skull, progressive mental aberration occurs in a previously sane patient who presents no other assignable cause for the intellectual malady. If there is pain or tenderness at the site of the injury, or if motor or sensory phenomena are found in the regions supplied by the nervous centres situated under the seat of injury, the demand for operation is much more imperative. I admit that cases reported as cured of acute maniacal symptoms by trephining are doubtful cures, because they might have recovered without operation. A number of chronic cases recovering after operation would afford much more conclusive evidence of the value of operative therapeutics.

Operation should be done before the pathological changes cause great imbecility. If pressure causes local degeneration or atrophy of the cerebral convolutions, it is imperative to remove that pressure as soon as it is evident that remedial drugs afford no relief and that the mental impairment is progressive. In an early part of this paper I have referred to a case seen by Dr. Mills and myself, in which I was prepared to trephine as soon as it was clearly established that the mental deterioration had no other assignable cause than the depressed fracture. Unfortunately the patient passed from observation.

It is impossible to fix any time; but progressive failure during three months would incline one to operate, and the same condition during six months would, I think, justify the surgeon in very strongly urging surgical treatment. On this point, however, I should like to hear the views of the Fellows of the Association.

In operating, all the depressed bone should be removed by

cutting away the intervening bone between the holes made by numerous applications of the trephine. The Hey's saw, gnawing forceps, or burr attached to the surgical engine will accomplish this. Guthrie[1] refers to a case of fracture, but not of insanity, in which twelve disks were removed before the depressed bone was properly elevated. Recovery followed.

Operation is not justifiable when there is evidence that some degree of insanity existed before the injury, nor when the patient has had, previous to the injury, insane delusions, not traumatic, from which he had recovered before the traumatism; nor when the character of the insanity is such that the pathological change is probably located in a region of the brain, distant from the seat of injury; nor when the insanity involves so many intellectual functions that general cerebral disease is clearly existent.

F. Operations for the removal of cerebral tumors have, as far as I know, been attempted only recently. The fear of approaching the brain with instruments deterred surgeons in a manner that seems unaccountable, when we recall the numerous instances of recovery from accidental brain injuries, that have been reported during the last fifty years. It is probable that most of those which recovered were of a character that insured efficient drainage, and that we did not have discernment enough to realize the true method by which fatal results were averted.

Primary cerebral tumors, which are not controlled by medicinal treatment, are usually single, seldom give rise to secondary deposits, are generally surrounded by an inflammatory zone of demarcation, and continue to increase in size until they destroy life. Therefore, when they are believed to be upon or in the cortex of the brain, or at least not deeply located in the cerebral substance, they should be removed by operation, unless they are in some such inaccessible and especially dangerous region as at the base of the brain.

Amidon[2] was one of the first, if not the first, to insist upon this method of dealing with cerebral tumors. A few months

[1] Gross's System of Surgery, vol. ii. p. 88. [2] Med. News, June 21, 1884.

ago, Godlee removed by enucleation a glioma the size of a walnut, situated a quarter of an inch below the surface of the ascending frontal convolution, and which had been located by Bennet.[1] For 20 days the patient did well; but he died on the 28th day from meningitis at the lower part of the wound, spreading down towards the base of the brain, on the same side, the whole of which was inflamed and covered with plastic lymph. The rest of the brain was normal. Amidon justly asks whether death might not have been due to the absence of antiseptic precautions, and the want of provision for drainage, especially as the galvanocautery was used to stop bleeding. I am not aware that the operator has definitely stated whether antiseptics were adopted and provision for drainage made.

This remarkable case of sagacious diagnosis by cerebral localization followed by a well-planned surgical operation is certainly very encouraging, and will doubtless be the incentive which will save many lives.

Multiple cerebral tumors, tumors of the base, and cerebral growths, secondary to neoplasm in other localities, should not be subjected to operation. If other circumstances obtain, and the symptoms are such as will localize the growth with reasonable certainty, excision should be attempted.

Sands records[2] a remarkable case in which Dr. Amidon diagnosed traumatic epilepsy in a patient who had struck the left parietal region against a sharp corner, but in which trephining showed no bone lesion. Death occurred in eight days, and a gummy tumor which could probably have been removed was found directly beneath the seat of trephining. No evidence of meningitis from the operation was apparent. This case may have been a potent factor in determining Dr. Amidon's well-known advocacy of the enucleation of cerebral tumors.

Dr. Charles K. Mills, who has made at least 25 to 30 autopsies in cases of brain tumor, of which number twelve have been

[1] Lancet, Dec. 20, 1884, and Jan. 3, 1885. See Amidon's discussion of its details in Annals of Surgery, March, 1885.

[2] Annals of Anatomy and Surgery, 1883, viii. p. 113.

published,[1] has called my attention to the fact that in several of them removal of the tumor would have been readily possible.

I give from his paper mentioned an abstract of two cases :—

Case IV.—Clinical history: Female, æt. 30. No history of causation. Headache continuous, sometimes agonizing. Percussion of head caused most pain in right parietal region. Vomiting when headache was most severe. Vertigo. Mind clear, but acted slowly; emotional. Spasms, beginning with twitching of fingers of left hand; most severe on left side, and especially in left arm. Upper as well as lower fibres of left facial nerve partially paralyzed; nearly complete paralysis of left arm; slight paralysis of left leg. Bowels and bladder partially paralyzed. Impaired sensibility of limbs of left side. Left patellar reflex diminished. Sight very imperfect. Choked disks. Hearing defective in right ear.

Pathological anatomy: Carcinoma, about one-and-a-half inch in diameter beneath and adherent to the pia mater of the convexity of the right hemisphere; the pia and dura mater were united by strong adhesions. The tumor involved the middle portion of the ascending parietal convolution and the upper part of the inferior parietal lobule, pushing aside the inter-parietal fissure. The anterior extremity of the tumor was about one-fifth of an inch back of the centre of the fissure of Rolando. On the inner side of the tumor the white matter of the brain was broken down.

Case V.—Clinical history: Female, æt. 38. History of syphilis. Blows on the head. Headache with agonizing paroxysms. Top and right side of head sensitive to percussion; and headaches severest in these regions. Vomiting. Vertigo. Great mental irritability. Severe left-sided spasm, beginning with twitching in left toes and foot. Partial paralysis of right leg and arm, most marked in leg. Hyperæsthesia. Impaired sight. Choked disk. Head temperature taken once; right parietal region, 97.2° F.; left parietal region, 96° F.

Pathological anatomy: Gumma. Attached to the fused membranes of the right convexity. Involved the upper one-fourth of ascending frontal, and a smaller segment of the ascending parietal convolution, crossing the fissure of Rolando at its upper extremity.

[1] Archives of Medicine, Aug. 1882.

A good example of strictly cortical lesion, involving only gray matter, and having only a very thin layer of softened tissue on its inner side. Microscopic examination of the optic nerves showed the appearance of choked disk, with probable ascending neuritis.

I have thus considered most of the topics which may be included under the head of cerebral surgery. In fractures, hemorrhage, and abscess, perforation of the skull will soon become quite common. In epilepsy, insanity, and tumor the profession will be more tardy in opening the skull. While I am sure that these conditions, in certain instances, can only be treated successfully by operative procedures, I believe also that other cerebral conditions, less well defined in pathology, such, for example, as incurable and intolerable headaches after injury, may in time be justifiably subjected to similar operations.

For such operations upon the skull and brain proper instruments should be at hand. For uncovering the centres of motion in searching for suspected lesions localized by symptoms, a trephine removing a disk three centimetres in diameter is none too large, for then both ascending convolutions and the intervening fissure of Rolando can be inspected with ease. A trephine with a cutting edge along only a half or two-thirds of its circumference, would, I think, be convenient at times for deepening a groove on one side of the disk, when the remaining portion of the circular groove has already completely cut through the bone in a thinner spot; and also for enlarging a trephine hole previously made. Dr. W. B. Hopkins has suggested a modification of the trephine which may be of value in this connection.[1] A probe with a large olive-shaped head and a light aluminium shaft, as recommended by Fluhrer, is certainly the proper exploring instrument in gunshot wounds of the brain. The head should, as he suggests, be placed so that the probe will be vertical and drop into the wound by its own weight. Aspirating needles should probably be made with a round end and a lateral opening like a catheter.

[1] Annals of Surgery, July, 1885.

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